Indian Society of Neuroradiology

ISNR News letter

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From the President's Desk

Dr. Mathew Cherian, President ISNR

The Neuroradiologist in the Age of Intelligence: Adaptation or Obsolescence?

The year 2026 represents a pivotal point for neuroradiology. We have transitioned from the "AI hype" phase to a period of clinical integration.

With more than 1,000 FDA-approved AI tools now accessible in radiology, the focus has shifted from questioning "Will AI replace us?" to considering "How can we effectively manage our new digital collaborators?"

Current Position: The Era of the Co-Pilot. In 2026, AI is no longer a mere novelty; it is thoroughly integrated into the neuroradiology workflow.

Its current influence is defined by three main components: Worklist Prioritization (Triage): AI algorithms now automatically review every incoming head CT and MRI for urgent "red flags" like intracranial hemorrhage (ICH), large vessel occlusions (LVO), and midline shift. These cases are promptly prioritized on the radiologist's worklist, significantly decreasing door-to-needle times for stroke patients.

Automated Quantification: Tasks that were once laborious—such as measuring multiple sclerosis (MS) lesion loads, calculating hippocampal volumes for dementia assessments, or monitoring tumor sizes—are now executed by AI with high accuracy. This has transformed the neuroradiologist's role from "data generator" to "data interpreter." Generative Reporting Assistants: Large Language Models (LLMs) incorporated into dictation software now create preliminary reports by

In This Issue

Scientific Summary

This issue highlights pediatric neuroradiology, presenting complex cases that challenge diagnostic paradigms. Key topics include the imaging spectrum of CNS leukemia, the rare association of Möbius sequence with syringomyelia, and dynamic MRI in MELAS. Reports also detail a unique case of NKH with congenital methemoglobinemia—where MRI provided a crucial "in-vivo" clue—and the endovascular management of a complex aneurysm in a 9-month- old infant.

"analyzing" the AI-detected findings and linking them with the patient's clinical history. The neuroradiologist now functions more as an editor, confirming the AI's findings rather than composing from scratch.

Future Directions: Precision and Prediction:

In the coming five years, AI is expected to evolve from merely assisting in diagnostics to becoming a formidable predictive tool.

We are heading towards "Zero-Click" Workflows: Future technologies will automatically retrieve previous exams, align them with current scans, and emphasize only the differences (such as a 2mm increase in a vestibular schwannoma), offering the neuroradiologist a pre-evaluated longitudinal comparison as soon as they access the study.

Opportunistic Screening: Standard brain imaging will be utilized to forecast future risks. AI will scrutinize "normal" scans to identify subtle indicators of vascular health or early signs of neurodegeneration long before clinical symptoms manifest, transforming neuroradiology into a proactive screening field.

Multimodal Fusion: AI will effortlessly combine imaging data with genomic and proteomic markers, delivering a "biopsy-like" diagnostic accuracy from non-invasive imaging.

Ensuring Relevance: Information Orchestrators

To ensure the current generation of neuroradiologists remains essential, a strategic shift in professional identity is necessary. We need to evolve from being mere "image readers" to becoming Information Orchestrators. The guiding principle for 2026 is that while AI excels at "narrow tasks" (like detecting a bleed), humans are better at "contextual synthesis" (such as understanding how that bleed fits into a patient's specific surgical history, comorbidities, and care objectives).

Lead the Governance Neuroradiologists should take the lead in validating, selecting, and overseeing AI tools. If we don't spearhead the adoption of these technologies, hospital IT departments or administrators might often sacrifice clinical subtleties.

Embrace the "Human-in-the-Loop" Model. Concentrate on the intricate 3D synthesis that AI still finds challenging. While AI can measure a lesion, it cannot yet handle the diagnostic uncertainty of rare "zebra" cases or effectively convey the significance of a finding to a worried family or a busy neurosurgeon.

Master the Tech Today's neuroradiologist should be as adept at discussing "data drift" and "algorithm bias" as they are at discussing "T2-FLAIR hyperintensities." Understanding AI's limitations—particularly its tendency for "hallucinations" in LLMs or false positives in detection—constitutes our new subspecialty expertise.

Reclaim the "Clinical" in Neuroradiology Use the time saved by AI automation to step out of the darkroom. Engage more thoroughly in multidisciplinary rounds, consult directly with patients, and offer higher-level consultative value to the clinical team.

Conclusion:

AI will not replace neuroradiologists, but neuroradiologists who use AI will certainly replace those who do not. The future belongs to those who can harmonize the speed of the machine with the wisdom of the clinician.

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From the Secretary's Desk

Dr. Ajay Kumar, Secretary ISNR

Pediatric Neuroradiology: Advancing Care for the Developing Brain

Pediatric neuroradiology demands a deep understanding of neurodevelopment, disease processes, and age-specific imaging characteristics. Alongside diagnostic imaging, pediatric neurointervention has emerged as a vital component of comprehensive care, offering minimally invasive solutions for complex cerebrovascular and spinal disorders. Together, these disciplines play a critical role in shaping lifelong neurological outcomes.

The pediatric nervous system undergoes continuous structural and functional changes, such as myelination, cortical maturation, and synaptic development influence normal imaging appearances and must be clearly distinguished from pathology. Early and precise diagnosis in conditions such as congenital malformations, metabolic disorders, hypoxic-ischemic injury, epilepsy, tumors, and neurodevelopmental syndromes can significantly improve long-term outcomes.

Pediatric neurointervention has expanded rapidly in recent years. Conditions such as arteriovenous malformations, aneurysms, vein of Galen malformations, acute ischemic stroke, vascular tumors, and spinal vascular lesions increasingly benefit from endovascular therapies.

Pediatric neuroradiology and neurointervention also faces significant challenges. Obtaining high-quality imaging in young children remains difficult due to limited cooperation and motion artifacts, often necessitating sedation or anesthesia. Radiation protection is another major concern, particularly in interventional procedures.

Interpretative and procedural challenges are equally demanding. Strengthening fellowship programs, mentorship, and institutional support is crucial for sustaining expertise.

Emotional and ethical challenges are inherent in pediatric practice.

The field continues to evolve through technological innovation and collaboration. Advances in fast MRI, functional imaging, artificial intelligence, low-dose fluoroscopy, and miniaturized devices promise to enhance diagnostic and interventional capabilities. Expanding global educational networks and multidisciplinary partnerships further strengthen training and knowledge sharing.

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Scientific Articles

From Leukemic Infiltration to ICANS: Imaging Spectrum of CNS Involvement in Pediatric Leukemia

Dr. Swathigha.S, Consultant Neuroradiologist, Kovai Medical Center, Coimbatore, India.

Introduction

Leukemia is the most common childhood malignancy, accounting for approximately 25 to 30 % of all pediatric cancers. Central nervous system involvement is frequent and may occur as either direct leukemic infiltration, indirect complications related to therapy or immunosuppression and increasingly as immunotherapy – related neurotoxicity, particularly with chimeric antigen receptor T-cell (CAR-T) therapy. Familiarity with this diverse imaging spectrum is essential for accurate diagnosis and timely management..

Direct CNS Manifestations

- Leukemic meningitis – linear and nodular leptomeningeal enhancement involving sulci, cisterns, folia and cranial nerves.
- Parenchymal leukemic infiltration – focal or diffuse lesions with T2/FLAIR hyperintensity, variable diffusion restriction and enhancement.
- Dural and calvarial disease – diffuse or focal dural thickening and enhancement, often associated with altered calvarial marrow signal.

Indirect CNS Manifestations:

- Vascular complications – intracranial hemorrhage due to thrombocytopenia, microbleeds related to DIC, arterial ischemic infarcts and cerebral venous sinus thrombosis.
- Opportunistic infections – secondary to prolonged immunosuppression, commonly manifesting as fungal abscesses or viral encephalitis.
- Methotrexate toxicity – acute - reversible areas of restricted diffusion in deep and periventricular white matter, chronic – cerebral volume loss.
- Radiation induced leukoencephalopathy – diffuse white matter signal changes with cerebral volume loss.
- Posterior Reversible Encephalopathy Syndrome (PRES) – associated with steroids, chemotherapy and hypertension and characterized by symmetric parieto-occipital predominant FLAIR hyperintensities.

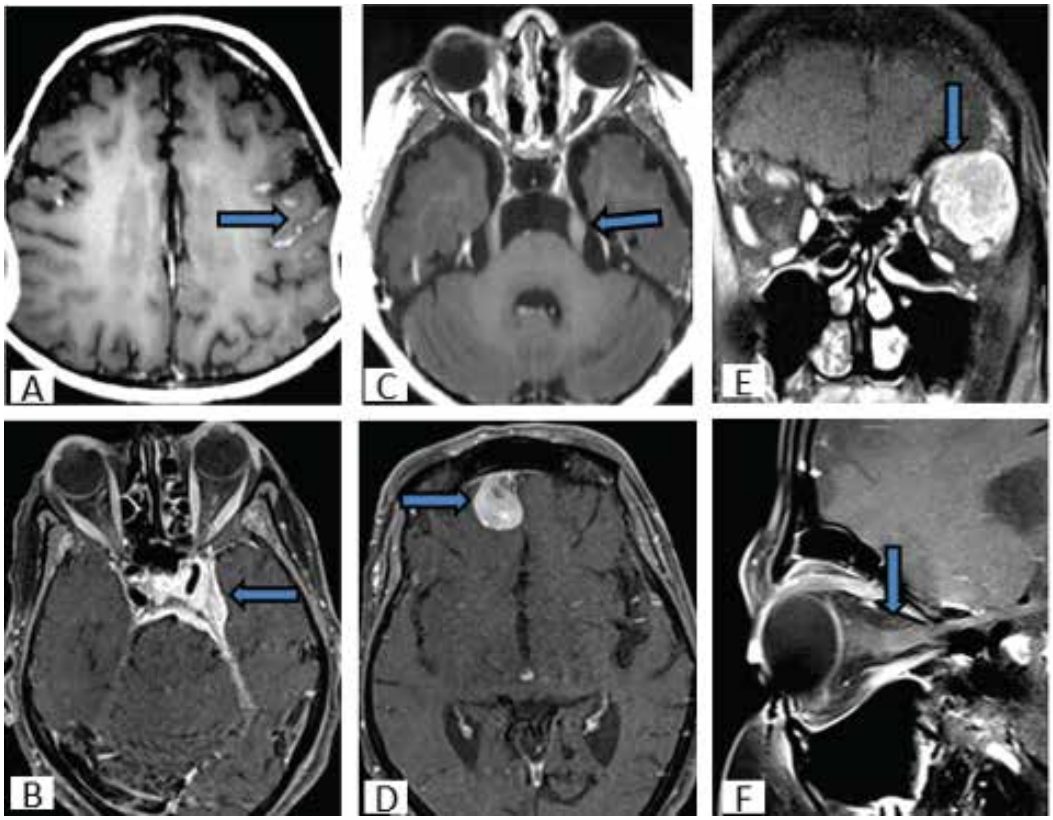
Immunotherapy Related CNS Complications:

- With the increasing utilization of CAR-T cell therapy for refractory hematological malignancies, Immune effector cell-associated neurotoxicity syndrome (ICANS) has emerged as an important complication.
- ICANS results from cytokine release, leading to activation of astrocytes and microglia, resultant inflammation and disruption of BBB.
- Imaging findings include focal or diffuse cerebral edema, white matter signal changes, splenial lesions of corpus callosum and microhemorrhages.
- Clinical manifestations range from delirium, aphasia, tremor, lethargy seizures and in severe cases may progress to coma and death.
- Neuroimaging maybe normal in early stages and radiological findings may lag behind clinical symptoms, underscoring the importance of clinic-radiological correlation.

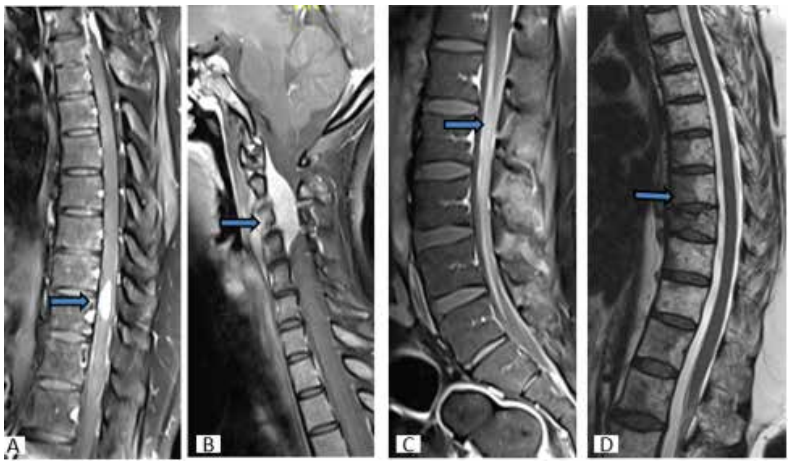
Conclusion:

A systematic imaging approach and awareness of the above mentioned entities play a crucial role in early diagnosis, differentiation from mimics and optimal management.

Direct Leukemic Manifestations:

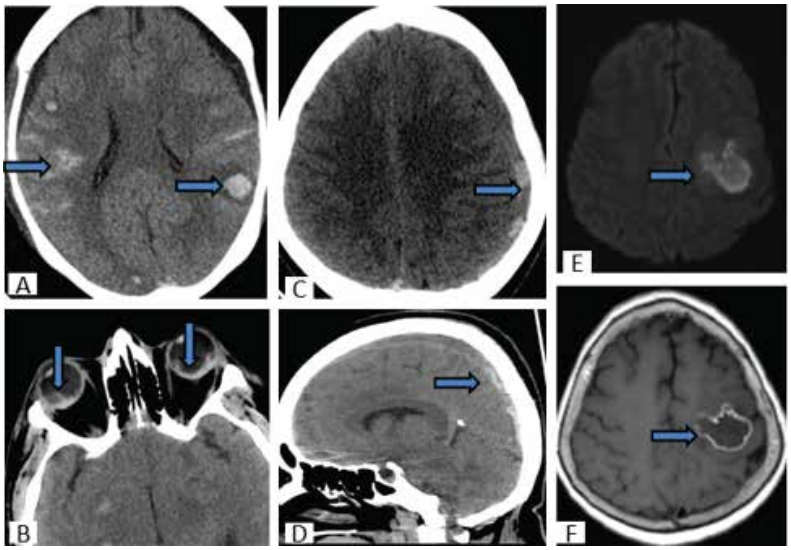


A- Nodular enhancing leptomeningeal deposits, B- Sheet like enhancing deposits along left tentorium and cavernous sinus, C- Cranial nerve thickening, D- Extra-axial dural based enhancing lesion, E- Left orbital xtraconal enhancing lesion and F- Left optic nerve thickening and enhancement.

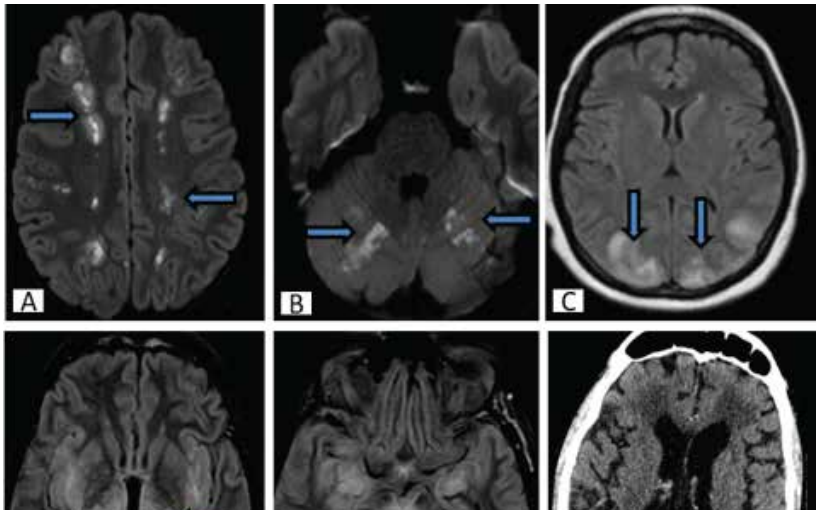


A- Intramedullary enhancing lesion, B- Extradural and paraspinal enhancing lesion, C- Cauda equine enhancement and D – Diffuse bone marrow infiltration.

Indirect Manifestations:

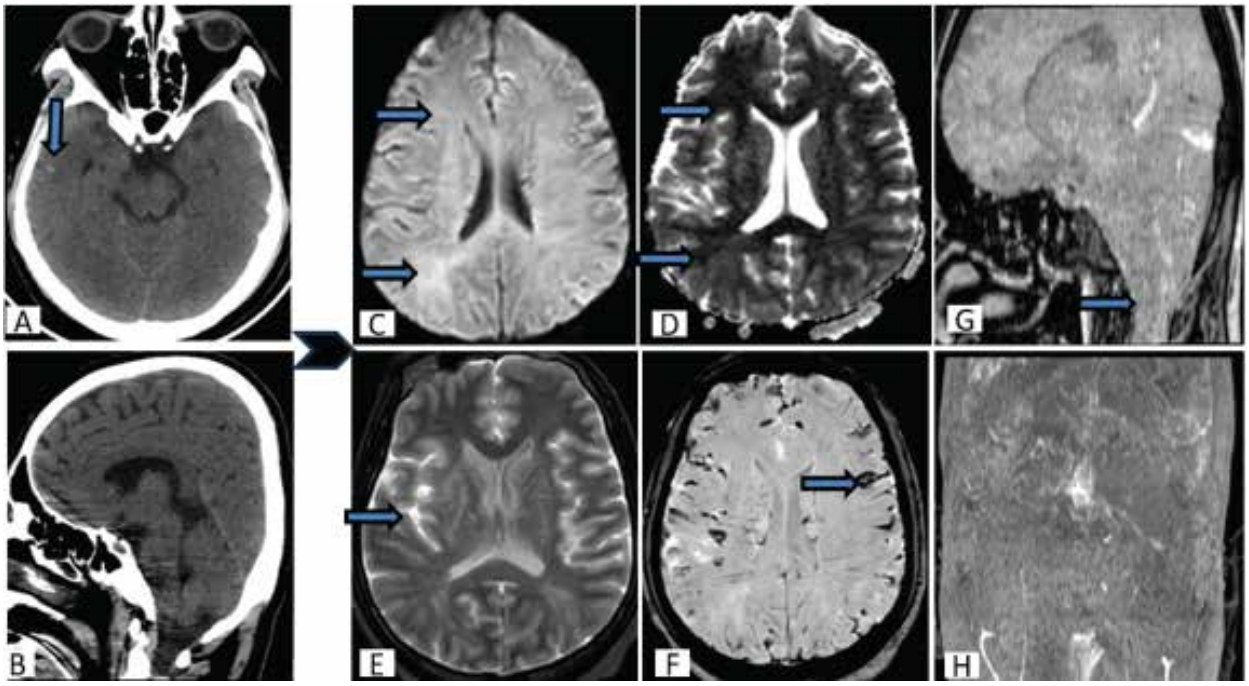


A- Diffuse SAH, multiple intraparenchymal hemorrhages and bilateral subdural effusion, B- Acute on chronic left cerebral convexity SDH, C- Bilateral vitreous hemorrhage, D- venous thrombosis of superior sagittal sinus, E and F – Cerebral abscess.



A&B – Bilateral watershed and cerebellar infarcts, C – Posterior Reversible Encephalopathy Syndrome(PRES), D&E – Acute toxic encephalopathy, F – Chronic radiation induced leukoencephalopathy with diffuse cerebral volume less.

Immune effector cell-associated neurotoxicity syndrome (ICANS):



A&B – Mild cerebral edema in CT (progressed within hours to death eventually) C&D – Diffuse cytotoxic edema with restricted diffusion in bilateral cerebral white matter, E – Diffuse sulcal FLAIR hyperintensities, F-Sulcal blooming secondary to congested veins, G - Tonsillar and brainstem herniation, H - Absent TOF flow in intracranial arteries.

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Möbius Sequence with Cervical Syringomyelia: Diagnostic Challenges and the Role of Targeted Brainstem Imaging

Dr. Nikita Mary Baby
Consultant Radiologist
MBBS, DNB (Radiology)
Fellowship in Paediatric Neuroradiology (SPIN)
Government Medical College, Kalamassery, Kochi, Kerala

Clinical History

A female neonate, the fourth child born of a non-consanguineous marriage, was referred for neuroimaging due to congenital cranial nerve dysfunction. Antenatal care was irregular, with no documented history of perinatal hypoxia or birth trauma.

From birth, the infant demonstrated **absent facial mimicking, incomplete eye closure, persistent open mouth posture, asymmetric lower lip movement, micrognathia with retrognathia, and swallowing dysfunction.** Clinical findings were suggestive of congenital facial nerve involvement. Ophthalmologic evaluation revealed retinal hemorrhages. A left parietal scalp swelling was noted clinically.

Imaging Findings

Initial MRI (1 week of age)

MRI of the brain with cervical spine screening was performed without contrast.

- The cerebral hemispheres, brainstem, cerebellum, and posterior fossa demonstrated normal morphology and signal intensity for age.
- There was **no evidence of Chiari malformation,** posterior fossa crowding, cerebellar tonsillar descent, hydrocephalus, or intracranial hemorrhage.
- **Micrognathia** with reduced mandibular size was noted.
- Head and neck imaging and interventions
- Cervical spine screening revealed a **cervical syrinx,** beginning approximately 1.1 cm below the cervicomedullary junction, measuring 2.6 cm in craniocaudal extent and 0.4 cm in anteroposterior diameter, with preserved craniocervical alignment and no cord signal abnormality. **A left parietal subgaleal hematoma** was present without intracranial extension.
- The presence of syringomyelia prompted initial diagnostic consideration of perinatal trauma and Chiari malformation, despite a normal posterior fossa. Interval follow-up imaging was advised.

Follow-up MRI with High-Resolution Brainstem Imaging (3 weeks of age)

Persistent cranial nerve dysfunction raised suspicion of **Möbius sequence,** prompting follow-up MRI with dedicated **high-resolution brainstem imaging,** including **3D CISS sequences.**

Key findings included:

- **Mild increase in the anteroposterior diameter of the mesencephalon**
- **Fusion of the quadrigeminal plate at the level of the inferior colliculi**
- **Mild tectal beaking**
- **V-shaped configuration of the fourth ventricle**
- No evidence of pontine hypoplasia, tegmental cap dysplasia, or middle cerebellar peduncle abnormalities
- Mild interval increase in the size of the cervical syrinx without cord edema
- Persistent subgaleal hematoma without intracranial extension

No supratentorial malformations were identified.

Diagnosis

Möbius sequence with associated brainstem dysmorphology and cervical syringomyelia.

Discussion

Möbius sequence is a rare congenital disorder characterized by non-progressive facial (VII) and abducens (VI) nerve palsies, frequently accompanied by craniofacial anomalies and variable involvement of other cranial nerves. Neuroimaging findings are heterogeneous and may be subtle or absent, particularly in early neonatal life..

A large AJNR imaging series by Ochoa-Escudero et al. demonstrated that Möbius sequence is frequently associated with brainstem and supratentorial developmental abnormalities, although none are universally present. Reported brainstem findings include increased anteroposterior midbrain diameter with shallow interpeduncular cisterns (≈31.6%), infratentorial arachnoid cysts (13.2%), and cerebellar vermian hypoplasia (5.3%).

In our patient, follow-up imaging demonstrated increased anteroposterior mesencephalic diameter, tectal beaking, quadrigeminal plate fusion, and a V-shaped fourth ventricle, aligning with previously described midbrain abnormalities in Möbius sequence. Importantly, no posterior fossa crowding or cerebellar vermian hypoplasia was present.

Supratentorial abnormalities reported in the AJNR cohort include **thalamic fusion, periventricular nodular heterotopia, ventriculomegaly, corpus callosum dysplasia, hippocampal malrotation, and arachnoid** cysts, each occurring in approximately 20–26% of patients. None of these supratentorial abnormalities were identified in this case, underscoring the wide **phenotypic and imaging spectrum** of Möbius sequence.

A distinctive and unusual feature of this case was the presence of cervical syringomyelia without Chiari malformation or posterior fossa abnormality. This association is rarely described in Möbius sequence and significantly influenced initial diagnostic reasoning, diverting attention toward traumatic and hindbrain etiologies and delaying recognition of an underlying brainstem developmental disorder.

Etiology and Differential Diagnosis

The most widely accepted etiology of Möbius sequence is hypoxic-ischemic injury to the developing brainstem, particularly the pontine tegmentum, where the abducens nuclei and facial nerve fibers traverse the facial colliculus.

MRI plays a critical role in excluding important mimickers, including:

Congenital fibrosis of the extraocular muscles (CFEOM), characterized by restrictive ophthalmoplegia without brainstem dysmorphology

Tubulinopathies, which demonstrate basal ganglia fusion due to internal capsule dysgenesis

Pontine tegmental cap dysplasia, showing pontine hypoplasia with a dorsal tegmental "cap" projecting into the fourth ventricle

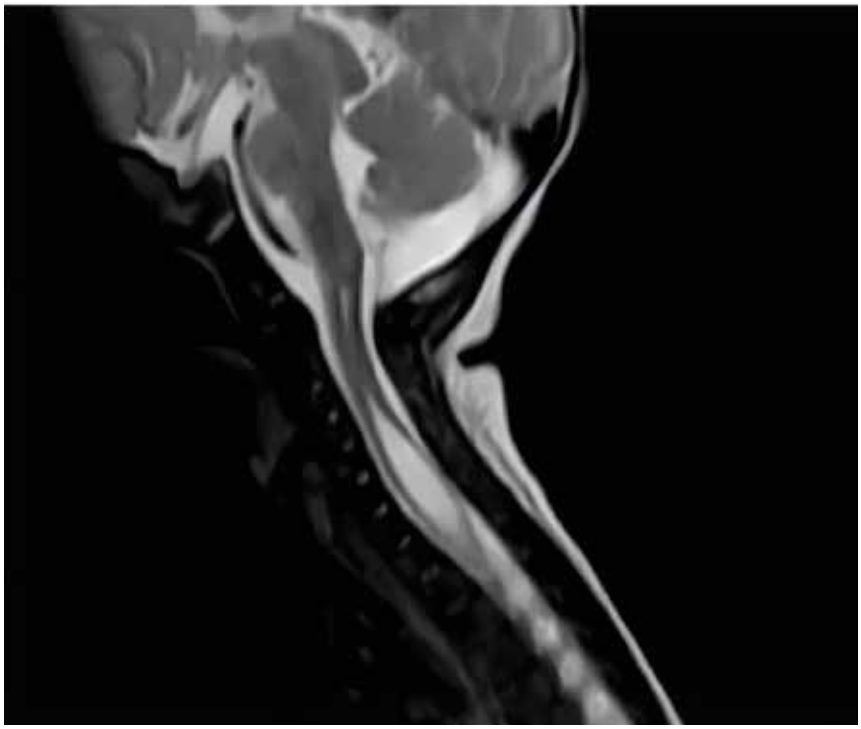
None of these imaging features were present in this patient.

Teaching Point

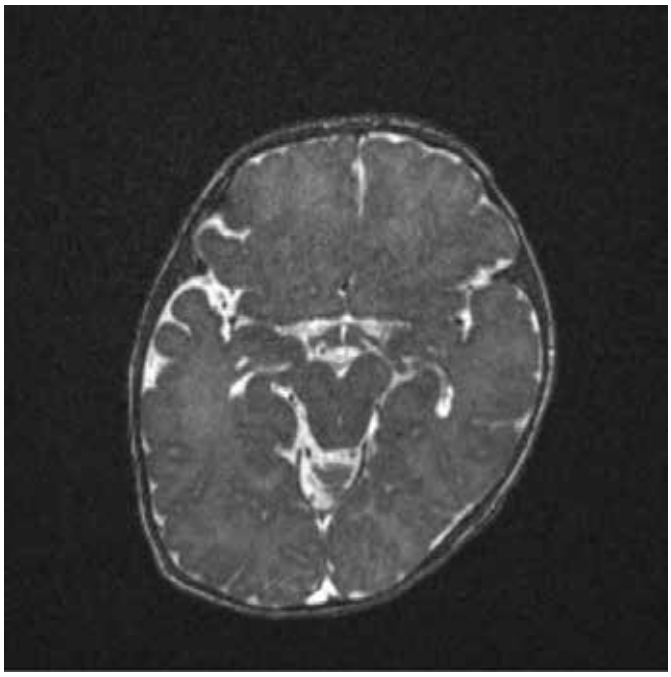
The identification of cervical syringomyelia in a neonate may divert diagnostic attention toward Chiari malformation or perinatal trauma and delay recognition of underlying brainstem developmental disorders. In neonates with persistent cranial nerve dysfunction, follow-up MRI with high-resolution brainstem sequences is essential, even when the posterior fossa appears normal.

Keywords

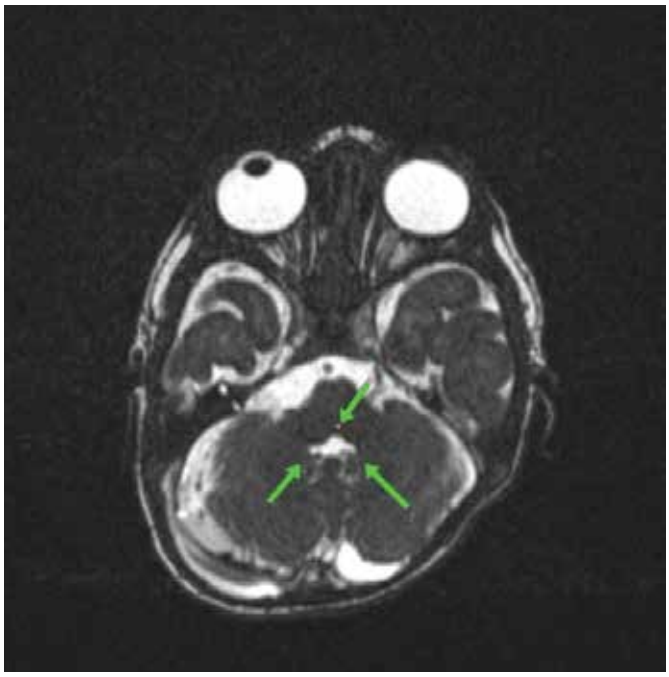
Möbius sequence; Neonatal MRI; Brainstem dysmorphology; Cervical syringomyelia; Cranial nerve palsy; 3D CISS



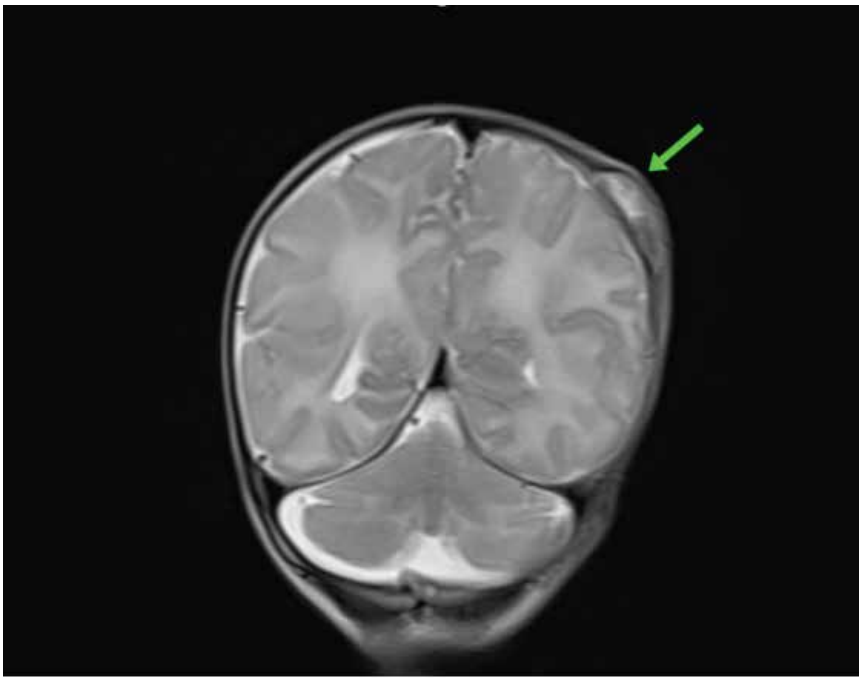
Sagittal T2-weighted MRI of the cervical spine demonstrates a syrinx extending inferior to the cervicomedullary junction.



Axial 3D CISS MRI at the level of the midbrain shows fusion of the inferior colliculi



Axial 3D CISS MRI at the level of the pons demonstrates flattening of the fourth ventricle with a central depression of the pontine tegmentum



Coronal T2-weighted image shows a left parietal subgaleal scalp collection, without intracranial involvement.

Clinical photograph demonstrates retrognathia with posteriorly positioned mandible, consistent with associated craniofacial involvement.

Clinical photograph demonstrates reduced facial animation with incomplete bilateral eye closure, asymmetric lower lip movement and micrognathia, reflecting congenital cranial nerve and craniofacial involvement.

Dynamic Diffusion MRI in Pediatric MELAS: Recognizing The "Shifting Lesion" Pattern

Dr. Nikita Mary Baby Consultant Radiologist MBBS, DNB (Radiology) Fellowship in Paediatric Neuroradiology (SPIN) Government Medical College, Kalamassery, Kochi, Kerala

Introduction

Mitochondrial encephalopathy, lactic acidosis, and stroke-like episodes (MELAS) is a rare but important metabolic disorder in pediatric neuroimaging. Early diagnosis remains challenging due to its variable clinical presentation and imaging patterns that often mimic ischemic or inflammatory conditions. This technical note highlights the role of dynamic MRI with serial diffusion-weighted imaging (DWI) in identifying evolving, non-vascular lesion patterns characteristic of pediatric MELAS, particularly in resource-limited settings

Clinical Context

A 6-month-old male infant presented with refractory seizures and encephalopathy following the initiation of complementary feeding. Initial laboratory evaluation showed severe lactic acidosis, raising suspicion of a metabolic disorder, though preliminary metabolic screening was negative. Neuroimaging was requested to evaluate ongoing neurological deterioration.

Imaging Technique and Protocol

MRI brain was performed with emphasis on:

Diffusion-weighted imaging (DWI) and ADC maps

T2-weighted and FLAIR sequences

Follow-up MRI with repeat DWI after short interval

MR spectroscopy focused on affected regions

Serial imaging was intentionally targeted rather than comprehensive, prioritizing sequences most sensitive to metabolic injury.

Imaging Findings

Initial MRI:

Multifocal cortica l and subcortical hyperintensities with diffusion restriction in bilateral parieto-occipital regions

Lesions crossed established vascular territories, inconsistent with arterial infarction

Follow-up MRI (Day 3):

Resolution of initial parieto-occipital lesions

- New diffusion restriction observed in:
  - Bilateral thalami
  - Basal ganglia
  - Dentate nuclei
  - Splenium of the corpus callosum

Evidence of a dynamic “shifting spread” pattern instead of lesion progression.

MR Spectroscopy:

Elevated lactate peak Increased glutamine/glutamate Reduced N-acetylaspartate (NAA)

These findingswere consistent with mitochondrial dysfunction, excitotoxic injury, and neuronal loss.

Technical Insight

The hallmark imaging feature in MELAS is not static stroke-like lesions, but temporal migration of diffusion abnormalities across non-vascular territories. Diffusion-weighted imaging is particularly sensitive in capturing these transient changes, often preceding or exceeding findings on conventional sequences.

MR spectroscopy provides critical biochemical corroboration, especially when genetic confirmation is delayed or unavailable.

Clinical–Radiological Correlation

The patient showed clinical improvement with resolution of refractory seizures, correlating with regression of diffusion abnormalities on follow-up MRI. This reinforces the importance of serial imaging rather than reliance on a single MRI study.

Practical Implications for Neuroradiology Practice

Stroke-like lesions outside vascular territories should prompt consideration of mitochondrial disorders

Interval MRIwith focused DWI is essential when initial findings are atypical

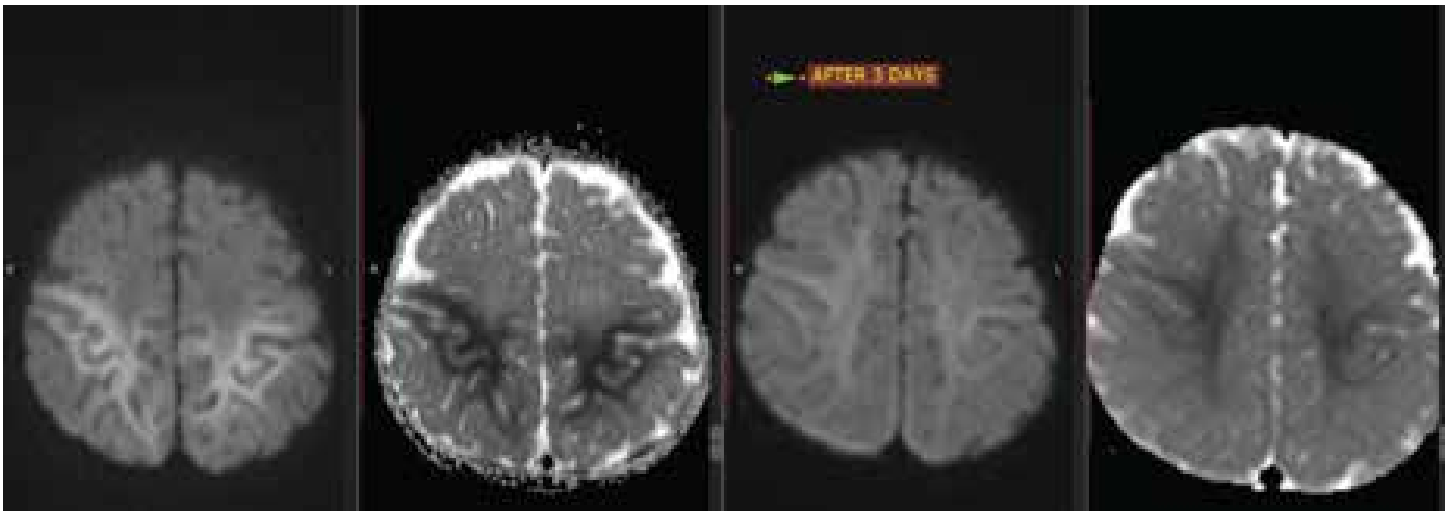
Targeted MRI protocols can provide early diagnostic confidence before genetic testing

This approach is particularly valuable in settings where advanced metabolic or genetic investigations

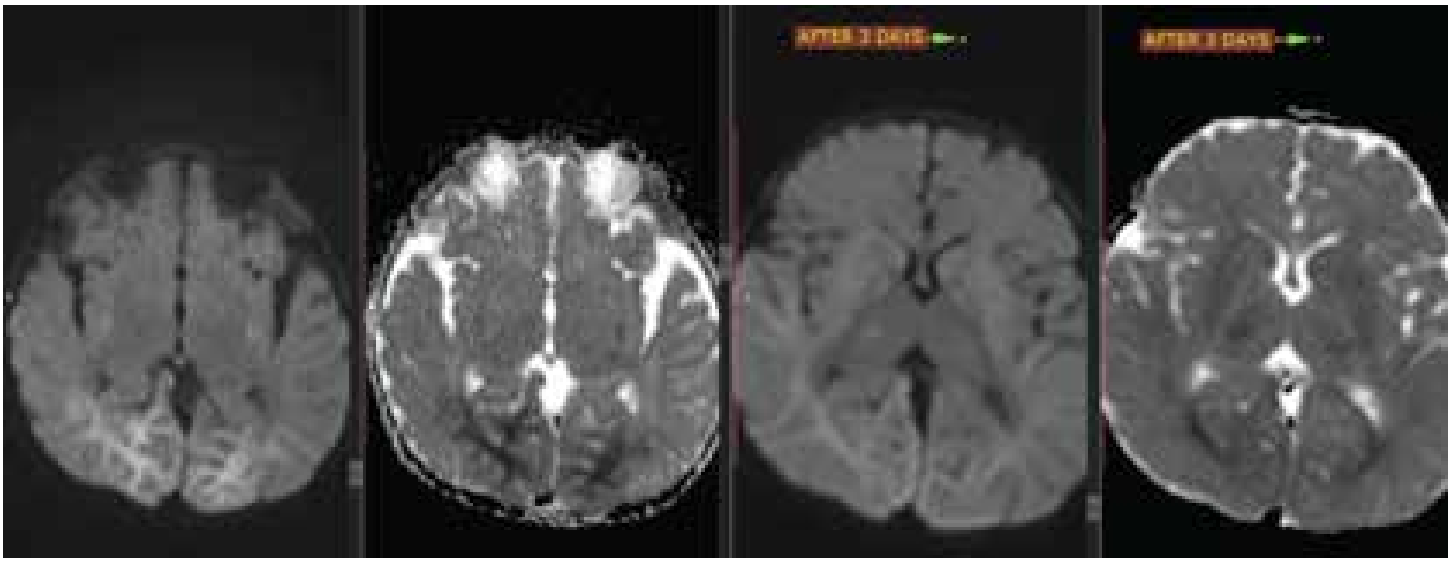
are not immediately accessible

Conclusion

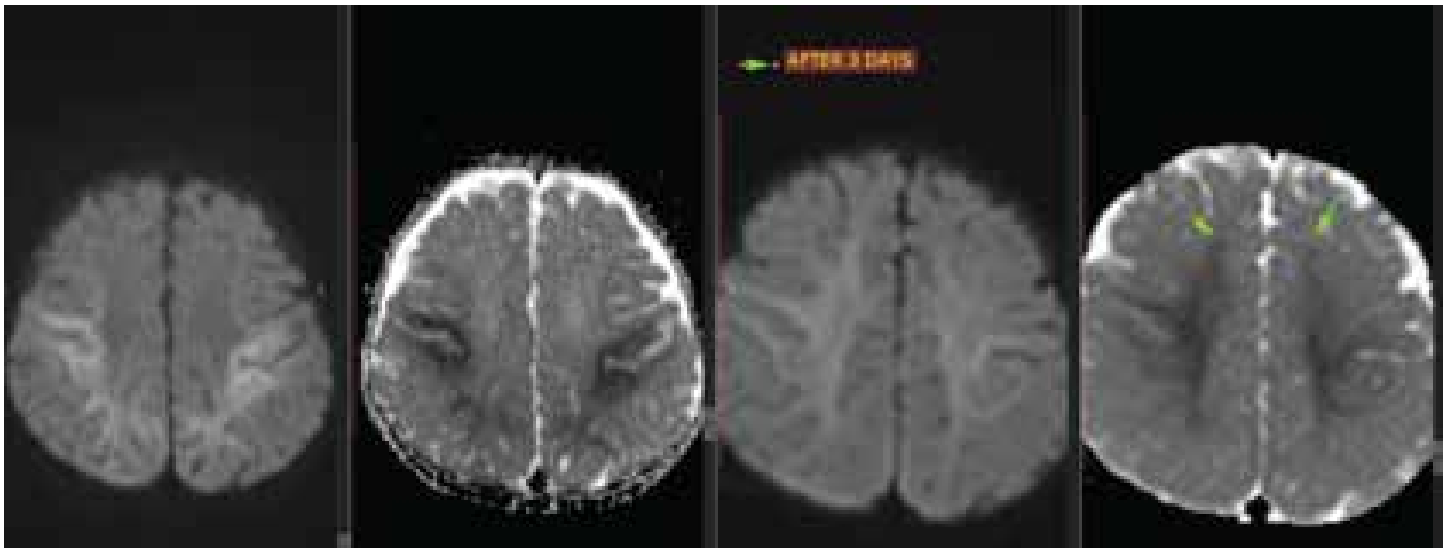
Dynamic MRI, especially repeated diffusion-weighted imaging combined with MR spectroscopy, plays a pivotal role in diagnosing pediatric MELAS. Recognition of evolving lesion patterns allows neuroradiologists to distinguish metabolic stroke mimics from true ischemia and initiate early management, even before genetic confirmation.



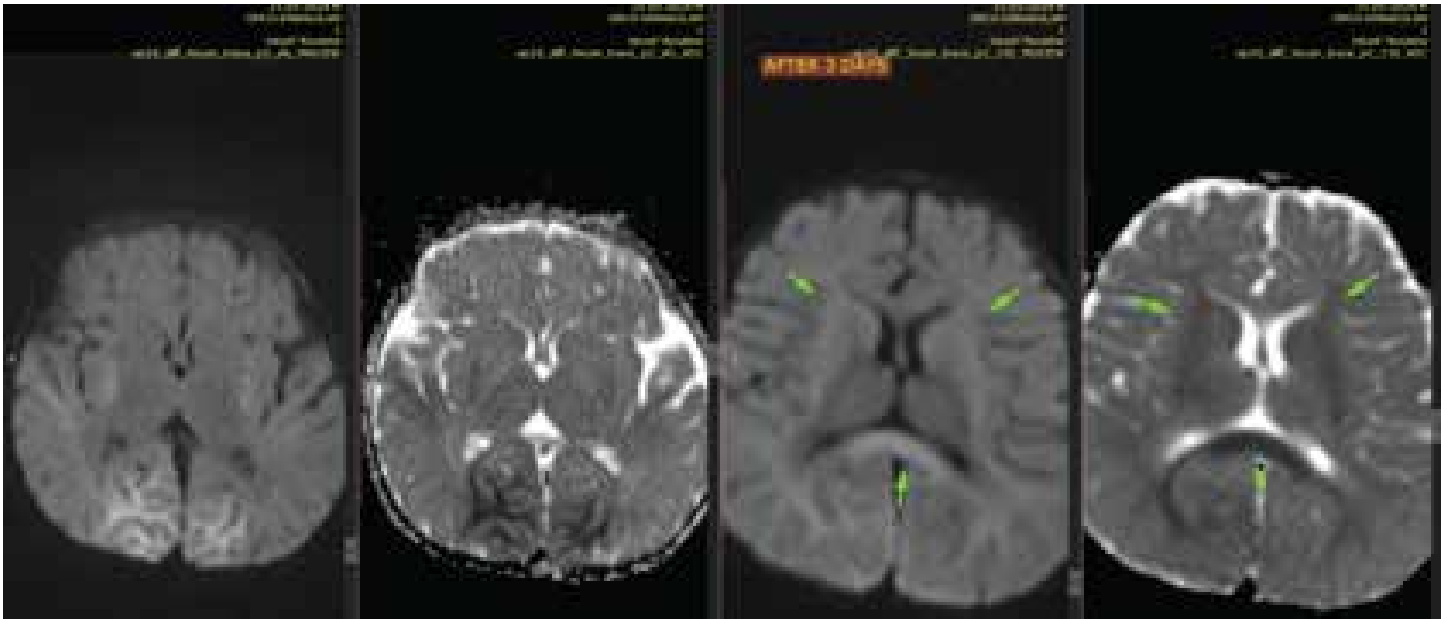
Follow-up MRI at 3 days demonstrates substantial resolution of previously noted cortical and subcortical edema, correlating with clinical improvement.



Follow-up MRI demonstrates interval reduction of cortical and subcortical edema in the bilateral parieto-occipital regions, correlating with clinical improvement.



Interval MRI reveals new areas of diffusion restriction in the deep white matter on follow-up imaging.



Follow-up MRI reveals new diffusion restriction in the basal ganglia, bilateral thalami, and splenium of the corpus callosum, reflecting evolving non-vascular lesion distribution.

The In Vivo Diagnostic Glow - A Rare Association of Non-Ketotic Hyperglycinemia with Congenital Methemoglobinemia – Pseudo enhancement sign

Authors: Dr. Anushuya Gunaseelan
Institution: Kovai Medical Center and Hospital, Coimbatore, India
Corresponding Author: Dr. Kannan Gunasekaran

Introduction

Neonatal encephalopathy with minimal biochemical derangement poses a significant diagnostic challenge. Inborn errors of metabolism (IEMs), particularly aminoacidopathies, are important causes. Non-ketotic hyperglycinemia (NKH) is a rare autosomal recessive disorder caused by defects in the glycine cleavage system, characterized by elevated glycine levels in plasma and cerebrospinal fluid with an increased CSF-to-plasma glycine ratio.

Methemoglobinemia is a rare condition in which hemoglobin iron is oxidized to the ferric state, impairing oxygen delivery. Methemoglobin causes T1 shortening on MRI, producing hyperintense signal within blood vessels on non-contrast T1-weighted images. Recognition of this phenomenon can provide a crucial imaging clue. We report a unique neonatal case of NKH with coexisting congenital methemoglobinemia, where MRI played a pivotal role in early diagnosis.

Clinical Profile

A term female neonate, second child of parents in a third-degree consanguineous marriage, was delivered by lower-segment cesarean section due to bad obstetric history. The first sibling had died 10 days after birth. There was a family history of neurodevelopmental delay in the father's brother.

The neonate presented within 12 hours of life with shrill cry, poor feeding, persistent cyanosis, low oxygen saturation refractory to oxygen therapy, and episodic bradycardia. By day 2 of life, there was progressive lethargy and excessive sleepiness. Neurological examination showed weak Moro reflex with preserved tone. Electroencephalography demonstrated a burst suppression pattern, suggesting severe encephalopathy.

On MR imaging, non-contrast T1-weighted images showed diffuse hyperintense signal within intracranial blood vessels and venous sinuses, mimicking post-contrast enhancement on a plain study ("pseudo-enhancement sign"). Other findings included Corpus callosal dysgenesis with Colpocephaly and widely spaced lateral ventricles. Symmetric FLAIR hyperintensity and diffusion restriction involving the posterior limb of the internal capsule, ventrolateral thalami, and corticospinal tracts were seen.

The pattern of tract-based involvement (PLIC and corticospinal tracts), callosal dysgenesis, and glycine peak on MR spectroscopy favored an aminoacidopathy, particularly Non-ketotic hyperglycinemia (NKH). The striking T1 hyperintense blood on non-contrast MRI raised suspicion of elevated methemoglobin levels, prompting targeted laboratory evaluation for methemoglobinemia.

Biochemical and Genetic Evaluation:

Blood gas analysis showed markedly elevated Methemoglobin level at 27.3%. Therapeutic trial of intravenous methylene blue administration resulted in reduction of methemoglobin level to 1.7%, confirming the diagnosis of congenital methemoglobinemia. Tandem mass spectrometry of blood confirmed grossly elevated glycine levels. Ultra-High Performance Liquid Chromatography showed plasma glycine levels of 3326 µmol/L (normal 166–330 µmol/L); CSF glycine levels of 345 µmol/L (normal 2–14 µmol/L); CSF-to-plasma glycine ratio of 0.10 (normal <0.03); Urine GC-MS (Gas Chromatography-Mass Spectrometry) showed no evidence of organic or dicarboxylic aciduria, diagnostic of NKH.

Genetic counseling was advised. The coexistence of two rare autosomal recessive conditions suggested independent pathogenic variants affecting glycine cleavage system genes and methemoglobin reductase pathways.

Discussion

Corpus callosal hypoplasia or dysgenesis is a well-recognized neuroimaging marker in several inborn errors of metabolism and serves as an important pattern-based clue in neonatal encephalopathy. Metabolic disorders associated with callosal abnormalities span multiple biochemical categories, including amino acid metabolism disorders such as non-ketotic hyperglycinemia, mitochondrial disorders including pyruvate dehydrogenase and fumarase deficiencies, and peroxisomal disorders, most notably Zellweger spectrum disorders and adrenoleukodystrophy. Other metabolic conditions, such as glutaric acidemia and congenital disorders of glycosylation, may also demonstrate callosal involvement.

In the present case, the coexistence of corpus callosal dysgenesis with symmetric tract-predominant white matter involvement—particularly affecting the posterior limb of the internal capsule and corticospinal tracts—significantly narrowed the differential diagnosis toward amino acid metabolism disorders, with non-ketotic hyperglycinemia being favored. The absence of severe metabolic acidosis, hyperammonemia, or ketonuria made organic acidemias and mitochondrial disorders less likely, while the lack of characteristic basal ganglia involvement or cortical malformations argued against peroxisomal disorders.

This pattern-based radiological assessment was further strengthened by MR spectroscopy demonstrating a persistent glycine peak at 3.5 ppm on both short- and long-echo-time spectra, a finding considered highly specific for non-ketotic hyperglycinemia. The constellation of corpus callosal dysgenesis, corticospinal tract involvement, and characteristic spectroscopic findings therefore allowed confident radiological narrowing of the diagnosis even prior to biochemical and genetic confirmation.

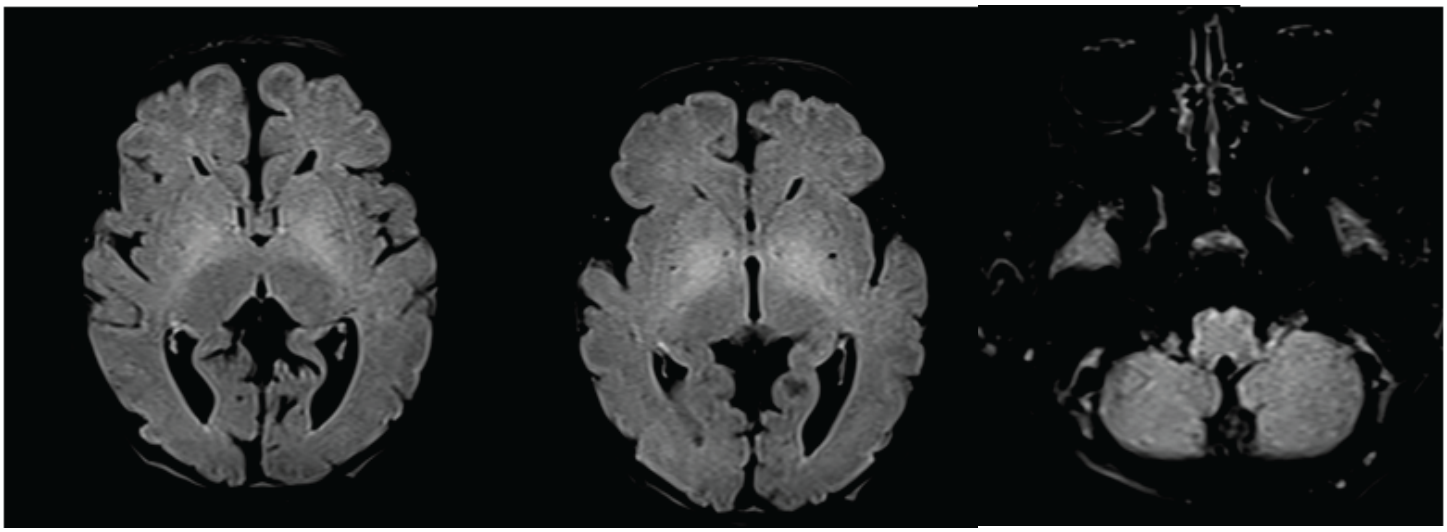
Methemoglobin is a well-recognized endogenous cause of T1 shortening due to its paramagnetic properties. Experimental and translational MRI studies have demonstrated a linear correlation between intravascular methemoglobin concentration and T1-weighted signal intensity, resulting in conspicuous hyperintense blood signal even in the absence of exogenous contrast agents. Recent literature has further explored

methemoglobin and related hemoglobin derivatives as potential endogenous MRI contrast agents, validating this imaging phenomenon.

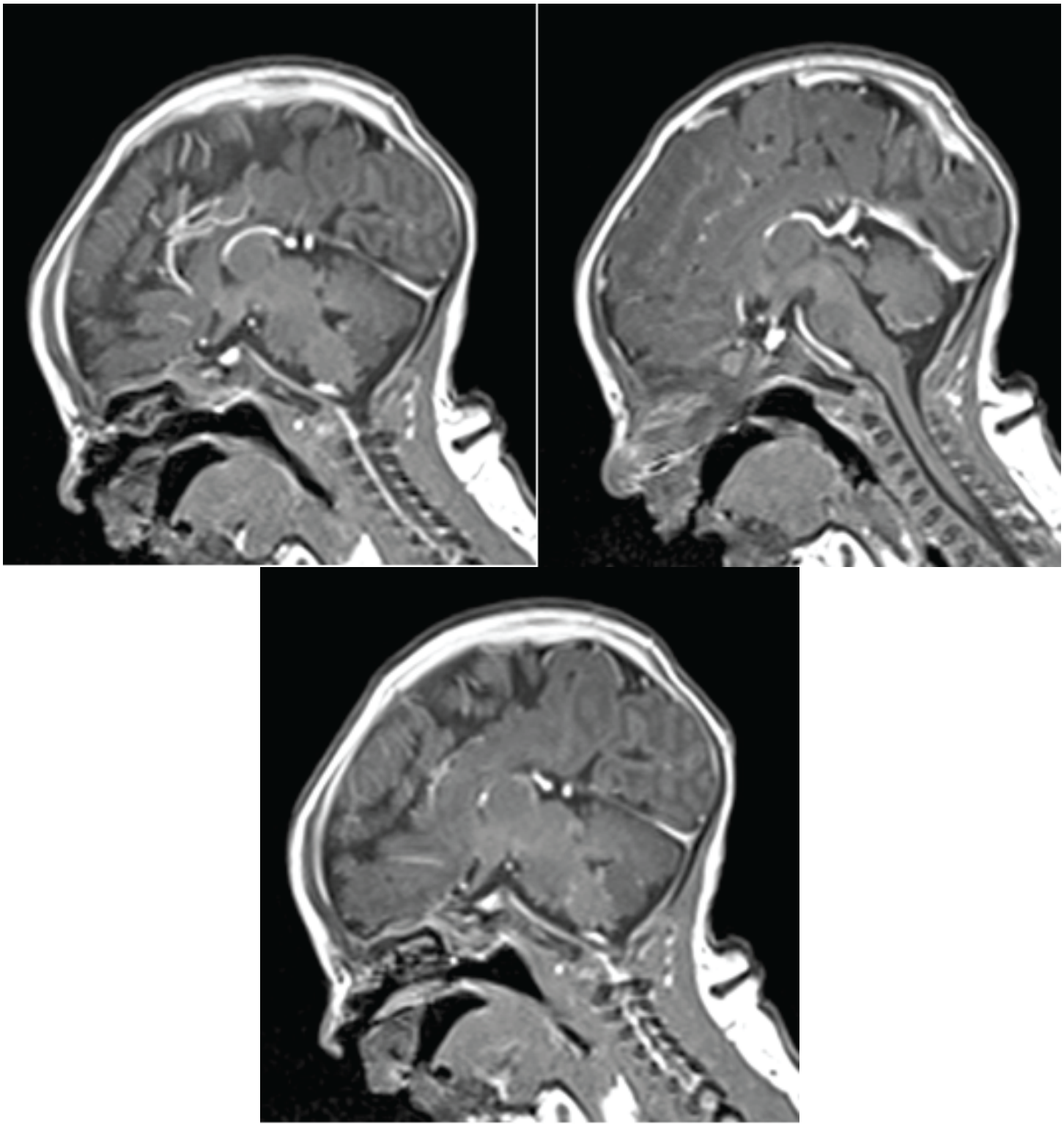
n our case, markedly elevated methemoglobin levels secondary to a pathogenic CYB5R3 mutation resulted in diffuse intravascular T1 hyperintensity on non-contrast MRI, mimicking post-contrast enhancement. Recognition of this pseudo-enhancement sign served as a critical diagnostic clue, prompting targeted biochemical testing and facilitating early identification of congenital methemoglobinemia. This case highlights the importance of integrating MRI physics, metabolic pattern recognition, and genetic correlation in the evaluation of neonatal encephalopathy.

Key Learning Points

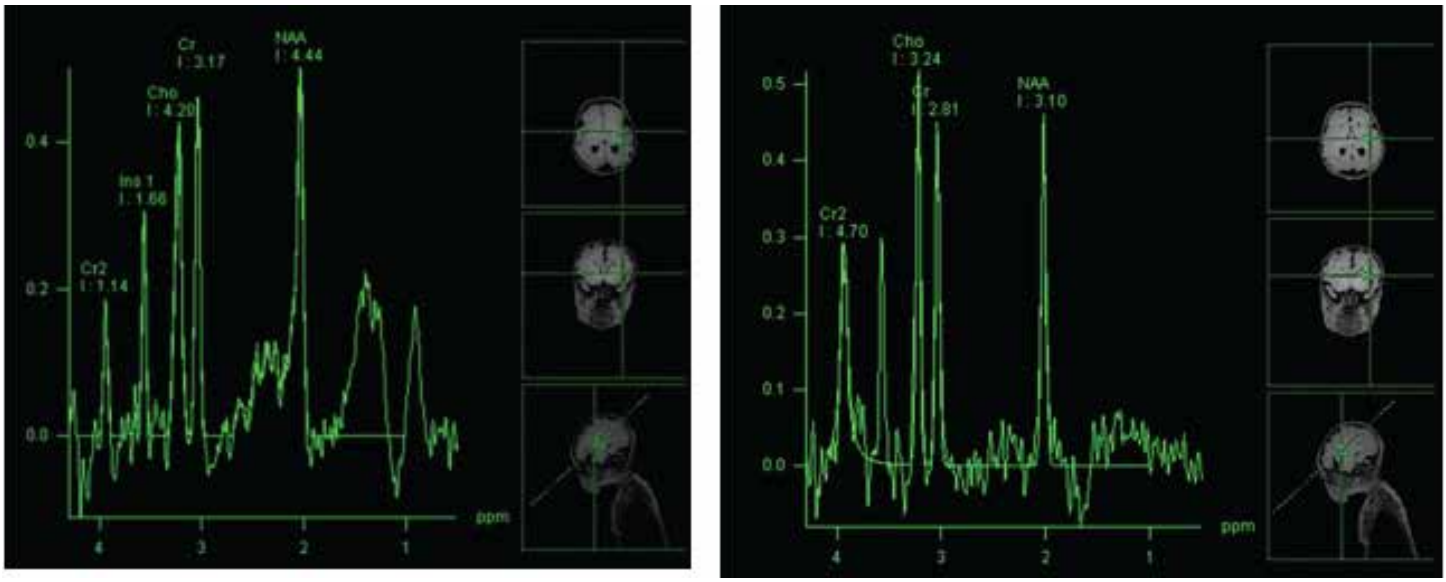
- Diffuse intravascular T1 hyperintensity on non-contrast MRI should prompt evaluation for methemoglobinemia.
- Tract-predominant white matter involvement with corpus callosal dysgenesis narrows the diagnosis toward amino acid metabolism disorders, particularly non-ketotic hyperglycinemia.
- MR spectroscopy demonstrating a glycine peak at 3.5 ppm in both long and short TE is highly specific for non-ketotic hyperglycinemia.
- MRI can function as an in-vivo diagnostic clue, guiding targeted biochemical and genetic testing in neonatal metabolic encephalopathy



Axial FLAIR images demonstrate symmetric hyperintensity along the corticospinal tracts involving the peri-rolandic cortex, posterior limb of the internal capsule, ventrolateral thalami, and anterior brainstem, and Colpocephaly.



Non-contrast axial T1-weighted MRI shows diffuse hyperintense signal within intracranial blood vessels and venous sinuses, mimicking post-contrast enhancement (“pseudo-enhancement sign”), secondary to elevated methemoglobin levels. Sagittal view reveals corpus callosal dysgenesis.



MR spectroscopy demonstrates a prominent glycine peak at 3.5 ppm on both short and long echo time spectra, diagnostic of non-ketotic hyperglycinemia.

RESULTS

LIKELY PATHOGENIC VARIANT CAUSATIVE OF THE REPORTED PHENOTYPE WAS DETECTED

Gene# (Transcript)	Location	Variant	Zygosity	Disease (OMIM)	Inheritance	Classification$
***CYB5R3* (-)** (ENST00000352397.10)	Exon 7	c.574C>T **(p.Arg192Cys)**	Homozygous	Methemoglobinemia due to deficiency of methemoglobin reductase (OMIM#250800)	Autosomal recessive	Likely Pathogenic **(PM1,PM2, PP3,PP5)**
***GLDC* (-)** (ENST00000321612.8)	Exon 6	c.818T>A **(p.Val273Glu)**	Homozygous	Glycine encephalopathy (OMIM#605899)	Autosomal recessive	Uncertain Significance **(PM2,PP3)**

No significant clinically relevant variants were detected in the mitochondrial genome.

Genetic analysis identifies a homozygous likely pathogenic variant in CYB5R3, confirming congenital methemoglobinemia, and a homozygous GLDC variant correlating with biochemically proven glycine encephalopathy.

Indian Society of Neuroradiology

Infant Intracranial Aneurysm and Endovascular Management

Dr. Vinayagamani Neuroendovascular & Interventional Radiology
Hannah Joseph Hospital, Madurai

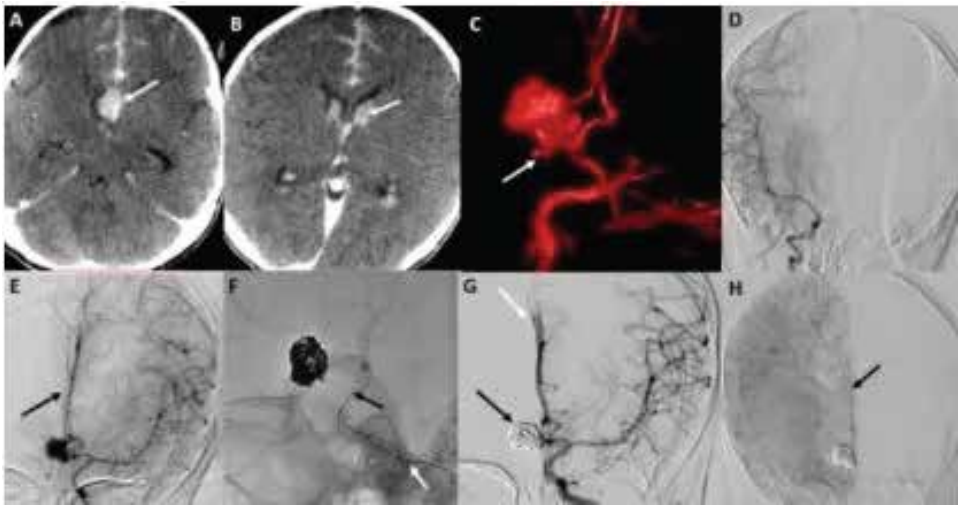
Dr. Uday Limaye is From Leelavthi Hospital

Case Summary:

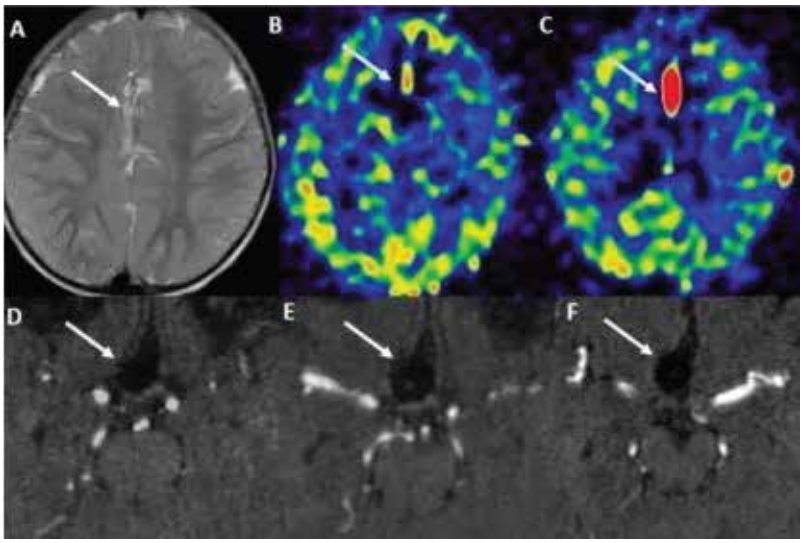
A previously healthy 9-month-old infant presented with recurrent seizures and altered sensorium (GCS 11). CT brain showed diffuse subarachnoid hemorrhage with intraventricular extension. Cerebral angiography revealed a large multilobulated saccular anterior communicating artery (ACom) aneurysm measuring 13 × 11 mm, associated with multiple small aneurysms along the right A2 ACA. There was severe right ACA vasospasm, with the ACA territory being maintained by robust MCA–ACA pial collaterals.

Considering the complex aneurysm morphology, severe vasospasm, strong collateral support, small vessel caliber, and the absence of validated pediatric antiplatelet protocols, a tailored endovascular strategy was adopted. The child underwent balloon-assisted coiling of the ruptured ACom aneurysm along with targeted parent artery occlusion of the diseased right A2 segment, achieving complete angiographic exclusion of the aneurysm complex. Reconstructive options such as stents or flow diversion were intentionally avoided.

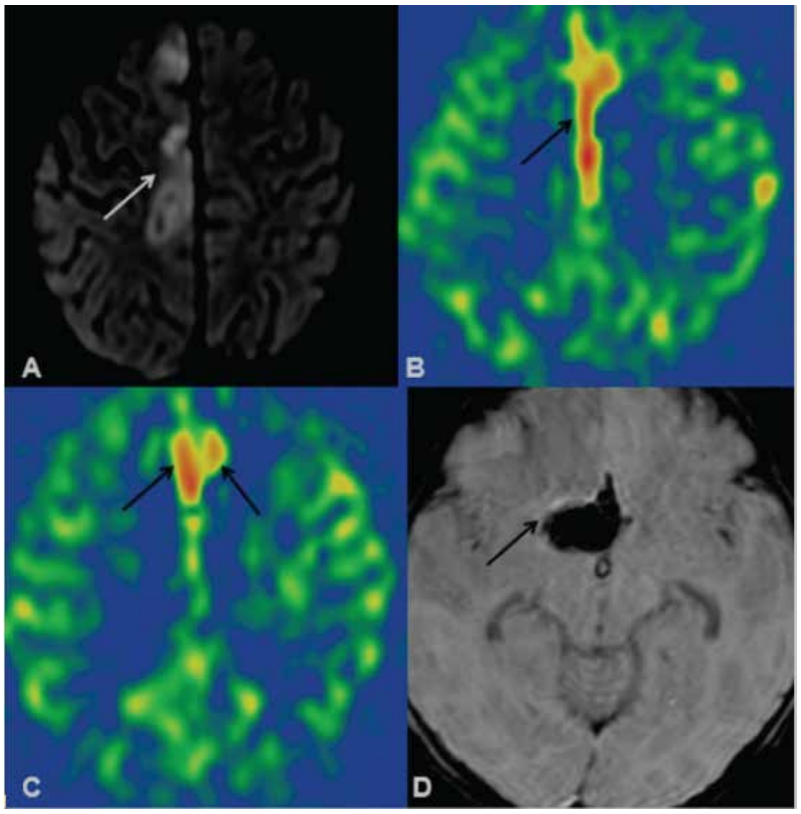
Post-procedure MRI showed patchy ACA territory infarcts related to vasospasm; however, the child made a complete neurological recovery without any gross deficit.



Large multilobulated ACom aneurysm.



6-month MRA: Complete occlusion



Follow-up: At 6 months, MRI/MRA demonstrated complete aneurysm occlusion with no recurrence, and the child showed normal growth and age-appropriate developmental milestones.

Indian Society of Neuroradiology

Report: ISNR Mid-Term CME 2025

Raipur, Chhattisgarh | October 11–12, 2025
Venue: AIIMS Raipur

Introduction

The **ISNR Mid-Term Continuing Medical Education (CME) 2025** was successfully conducted on **October 11 and 12, 2025 at AIIMS Raipur, Chhattisgarh,** under the aegis of the **Indian Society of Neuroradiology (ISNR).** The CME served as a high-quality academic platform for **neuroradiologists, radiologists, neuroradiology fellows, neurosurgery trainees, and early-career interventionists** to update their knowledge and skills in both **diagnostic and interventional neuroradiology.**

The event witnessed **enthusiastic national participation with nearly 200 registrations** and **around 50 scientific entries for oral and paper presentations,** reflecting strong academic engagement in neuroradiology across India. The CME featured **eminent national faculty,** making it a major academic forum for learning, professional exchange, and advancement of neuroradiology practice in the country.

Day 1 – October 11, 2025

The first day of the CME commenced with **live diagnostic and interventional neuroradiology workshops.** The workshops attracted strong trainee participation, with **around 30 entries for the Diagnostic Neuroradiology Workshop,** along with active participation in the interventional workshop.

Diagnostic Neuroradiology Workshop

The Diagnostic Neuroradiology Workshop, conducted by Dr. Shriram Varadharajan and Dr. Aarthi Deepesh, included advanced modules on:

- **Perfusion imaging**
- **Task-based functional MRI (fMRI)**
- **Diffusion tensor imaging (DTI) and tractography**
- **Cerebrospinal fluid (CSF) flow studies**

These sessions provided in-depth understanding of the role of advanced neuroimaging in neuro-oncology, presurgical planning, stroke evaluation, and disorders of CSF dynamics.

Interventional Neuroradiology Workshop

A major highlight of the interventional workshop was a **live demonstration of cerebellar arteriovenous malformation (AVM) embolization, performed by Dr. Nihar Vijay Kathrani and elucidated by Dr. Vivek Lanka.** The live procedure offered participants valuable real-time insights into **case selection, catheter navigation, embolic agent selection, and intraprocedural imaging guidance** in a complex neurovascular intervention.

Inaugural Session

The **formal inaugural ceremony** was held after the morning workshops and marked the official commencement of the **ISNR Mid-Term CME 2025. The session was attended by ISNR office bearers, senior** faculty, and the leadership of AIIMS Raipur, who emphasized the importance of **structured neuroradiology training, innovation in imaging, and the expanding role of neuro-interventional** services in India.

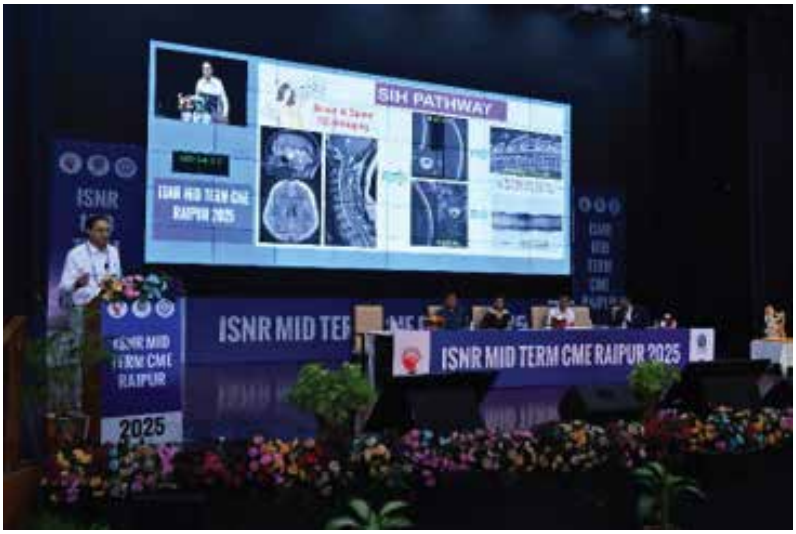
The dignitaries congratulated the organizing committee for creating a strong academic platform that brought together **leading national experts and young trainees** for meaningful exchange of knowledge and improvement of patient care. The inaugural session set a **high academic and professional tone** for the two-day conference.

Day 1 Scientific Sessions

Following the inauguration, the scientific program focused on **core neurovascular and CSF-related disorders,** with lectures on:

- **Wide-neck bifurcation aneurysms (WBNA)**
- **Vascular malformations**
- **Basics of carotid artery stenting**
- **The pressure puzzle: SIH and IIH – CSF leaks, blocks, and balance**
- **Spectrum of SIH and IIH**

These sessions provided **evidence-based, clinically relevant insights** into complex neurovascular conditions and CSF pressure disorders.



The first day concluded with a **gala dinner held on the evening of October 11, 2025,** which provided an excellent opportunity for **faculty, delegates, and trainees to interact informally, strengthen professional relationships, and foster collaboration** in the field of neuroradiology.

Day 2 – October 12, 2025

The second day was dedicated to **academic presentations and advanced scientific discussions.** The day began with **oral and paper presentations** by residents and fellows, showcasing **original research, interesting case series, and novel imaging findings** in both diagnostic and interventional neuroradiology. These presentations were critically evaluated by expert faculty, promoting **scientific rigor and academic excellence.**

This was followed by scientific sessions covering:

- **Acute stroke imaging and intervention**
- **Tumor imaging and neuro-oncology**
- **Neuroimmunology and white-matter diseases**
- **Head and neck imaging and interventions**
- **Neuroinfections and neurodegenerative disorders**
- **Advanced and emerging topics included: Artificial intelligence in neuroradiology, Neuroimaging in epilepsy, Calvarial and spinal bony lesions.**

The academic program concluded with a highly engaging **Neuroradiology Quiz,** followed by the **valedictory session and prize distribution.**

Organizing and Scientific Committees

ISNR Office Bearers

- **President: Dr. Mathew Cherian**
- **Vice President: Dr. Hirdesh Sahni**
- **General Secretary: Dr. Ajay Kumar**
- **Joint Secretary: Dr. Deb Baruah**
- **Treasurer: Dr. Sameer Vyas**

ISNR Central Executive Committee

Dr. Arvinda H.R., Dr. Nihar Vijay Kathrani, Dr. Rasmiranjan Padhi, Dr. Roopa S., Dr. Shriram V., Dr. Somnath P., Dr. Uday Bhanu, Dr. Vivek Singh

Conference Organizing Committee

Patron

Lt. Gen. Dr. Ashok Jindal (Retd.), Executive Director & CEO, AIIMS Raipur

Co-Patrons

Dr. Eli Mohapatra (Dean Academics)
Dr. Abhilash Galhotra (Dean Research)
Dr. Avinash Ingle (Dean Examinations)
Dr. Krishnadutt Chavali (Dean Students' Welfare)
Dr. Renu Rajguru (Medical Superintendent)

Organizing Chairperson

Dr. Narendra Kuber Bodhey

Organizing Secretaries

Dr. C. D. Sahu
Dr. Nihar Vijay Kathrani

Treasurers

Dr. Richa Singh Chauhan
Dr. Saroj Kumar Pati

Scientific Committee

Chairperson: Dr. Manish Kumar
Members: Dr. Anand Bansal, Dr. Nilesh Gupta, Dr. Prashant Pote, Dr. Shreyas Jaiswal

Conclusion

The **ISNR Mid-Term CME 2025, held at AIIMS Raipur,** was a **highly successful and academically enriching national event.** With **nearly 200 delegates, hands-on diagnostic and interventional workshops, complex live neuro-interventional demonstrations, and strong resident participation,** the CME significantly strengthened **neuroradiology education and clinical practice in India.**

The event reaffirmed ISNR's commitment to **academic excellence, innovation, and high-quality neuroimaging and neuro-interventional care.**

Indian Society of Neuroradiology
ISNR

Young Neuroradiologists Forum (YNRF) Update

YNRF, the youth wing of ISNR, was officially inaugurated in October 2025 at the Mid-Term CME in Raipur having a convener and eight other members as follows.

1. Dr SHRIRAM VARADHARAJAN- Convener
2. Chirag Jain, Delhi -member
3. Madan m Gupta, Jaipur -member
4. Vinay Goel, Delhi -member
5. Nirmalaya Ray, Kolkata -member
6. Arpita Sahu, Mumbai -member
7. Bharat Hosur, Pune -member
8. Sabha Ahmad, Bangalore -member
9. Shashank Raj, Delhi -member

Monthly webinars, moderated by the Academic Coordinator, have covered diverse topics led by eminent faculties, both national and international under the auspices of the past, current presidents, secretary and the rest of the current ISNR EC.

Webinar Topics

- Headache awareness and intracranial hypertension
- Holistic stroke continuum beyond routine care
- Pediatric neuroimaging: leukodystrophies
- CNS infections (AIDS Awareness Month)
- AI applications in neuroradiology
- Women's health: interventional radiology panel and fetal MRI
- Endovascular stroke trials: current perspectives and future directions
- Evolution of endovascular aneurysm treatment
- Chronic SDH-review and embolization

Webinar Topics

The first member cohort has been formed with a mix of diagnostic and interventional backgrounds. The first online meeting was convened and discussed future initiatives.

Stay tuned for all the updates on @ISNRClinics academic social media handle of YNRF.

Indian Society of Neuroradiology

Upcoming Annual Conference

27th Annual Conference of Indian Society of Neuro-radiology (ISNR) is being organised from 27th to 29th March 2026 by Department of Neuroimaging and Interventional Radiology, PGIMER, Chandigarh. The conference will be held in beautiful city of Chandigarh at Hyatt Regency.

Along with distinguished national faculties, various well known international faculties are going to join the conference as well, including- Dr René Chapot (Germany), Dr Ajit Puri (USA), Dr Tufail Patankar (UK), Dr Mario Martínez-Galdámez (Spain), Dr Bruno Parente (Brazil), Dr Ian Rennie (UK), Dr Youngjin Jung (South Korea), Dr Ajay Malhotra (USA), Dr Dhairya Lakhani (USA), Dr Gaurav Saigal (USA), Dr Karuna Shekdar (USA), Dr Kish Mankad (UK), Dr Mark Foley (USA), Dr Neetu Soni (USA), Dr Pradeep Krishnan (Canada), Dr Rajan Jain (USA), Dr Rupa Radhakrishnan (USA), Dr Sandeep Bhuta (Australia), Dr Shubh Biswas (UK), Dr Srikala Narayanan (USA), Dr Tilak Das (UK), Dr Vijay Sawlani (UK).

Registration can be done at https://isnr2026.com/index.html . Conference highlights includes Workshops for Interventions and Advance Imaging, CPCR / Airway Workshops, RPCs (Radiologico – Pathological Correlations) Sessions, Interactive Quizzes, Various Panel Discussions, Fast Track Onsite ISNR Membership options etc.

Beloved Radial: An Optimistic Love Letter

Vikas Bhatia, MD, DM Additional Professor, PGIMER, Chandigarh

"The Intervention Angiosuite" Acrylic on Canvas. Vikas Bhatia 2024

Beloved Radial,

I was comfortable with Femoral, my long-term friend,
With a wide lumen and history on which I could depend.

Then you entered my practice, slim and tender,
Changing my outlook as a new contender.

You showed me spasms, you were easily irritable,
Yet with time and patience, you became reliable.

You ease the procedure, with complications so few,
Discharging patients early, a victory for the crew.

Femoral remains a friend, and that stays true,
But Radial, my focus is now also on you.