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Indian Academy of Neurology

# Advances in Stroke Rehabilitation

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CHAPTER 10

# Stroke in Children and Rehabilitation Measures

*PA Mohammed Kunju*


## ABSTRACT

Sudden occlusion or rupture of cerebral arteries or veins, resulting in focal cerebral damage and clinical neurological deficits is defined as stroke.[1] And World Health Organization (WHO) defines it as a "a clinical syndrome of rapidly developing focal or global disturbance of brain function lasting 24 hours or leading to death with no obvious nonvascular cause".[2] Clinical features of stroke in children vary considerably from adult stroke. Similarly etiologies are also very much different. In this chapter, these features are highlighted with special emphasis on arteriopathic stroke and vasculitis-related stroke. Rehabilitative methods like physiotherapy, speech, and occupation therapy techniques are also highlighted. Role of botulinum toxin (BTX) in pediatric stroke is described along with the technique of injection


**Keywords:** Occlusion, WHO, Global disturbance, Clinical features, Botulinum.

## INTRODUCTION

Stroke is defined as sudden occlusion or rupture of cerebral arteries or veins, resulting in focal cerebral damage and clinical neurological deficits. World Health Organization (WHO) defines it as a "a clinical syndrome of rapidly developing focal or global disturbance of brain function lasting 24 hours or leading to death with no obvious nonvascular cause". Stroke in the young occurs below 40 years of age. One-fourth of this is in children. The causes of stroke are also different—nonatherosclerotic vasculopathies, abnormalities of coagulation, heart disease, and sickle cell anemia dominate the list in pediatric population. Different presentations of arteriopathic stroke like transient cerebral arteriopathy is increasingly seen in children. Among them varicella zoster arteriopathy is a leading cause. Along with various heart diseases, vasculitis, both primary central nervous system (CNS) angiitis and systemic vasculitis, is an etiology to be described in detail.

Rehabilitative techniques including physio, occupational, and speech therapy is highly indicated and may have to be given in a prolonged manner.

## SPECIAL FEATURES OF STROKE IN CHILDREN

Stroke in young occurs below 40 years of age. One-fourth of this is in children. Incidence rates appear to be increasing from 1/100,000 to 2.5–2.7 per 100,000 children.

- The causes of stroke are also different—nonatherosclerotic vasculopathies,

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abnormalities of coagulation, heart disease, and sickle cell anemia dominate the list.[3]

- Seizures are also more common in childhood stroke. Focal seizures are often the presenting symptom in neonatal stroke.
- Unlike in adults where arterial thrombosis is common due to small striate artery lipohyalinosis children get stroke due to large artery occlusion. Cardiac causes are equal to other causes.
- Children have hemorrhagic strokes as often as they have ischemic strokes, while adults are more likely to have ischemic strokes.

## CLASSIFICATION OF STROKE

Stroke can be ischemic or hemorrhagic. Ischemic stroke is again divided into arterial ischemic stroke (AIS) and venous ischemic stroke **(Flowchart 1)**.

## SIGNIFICANCE OF ISCHEMIC PENUMBRA

In every infarct, there is a central core of total ischemia and necrosis, which is irreversible. This area is surrounded by a zone of borderline ischemic tissue, the ischemic penumbra. Ischemia, in the penumbra, causes dysfunction due to ionic and metabolic dysfunction but is not severe enough to result in structural damage.

## CLINICAL PRESENTATIONS OF STROKE

Stroke should be considered when there is sudden onset of weakness or loss of function like aphasia occurring without fever **(Box 1)**. Young children—subtler, nonspecific symptoms like irritability, confusion, headache, and behavioral changes can be the presentations. Symptoms involving a particular arterial territory are described in **Table 1**.

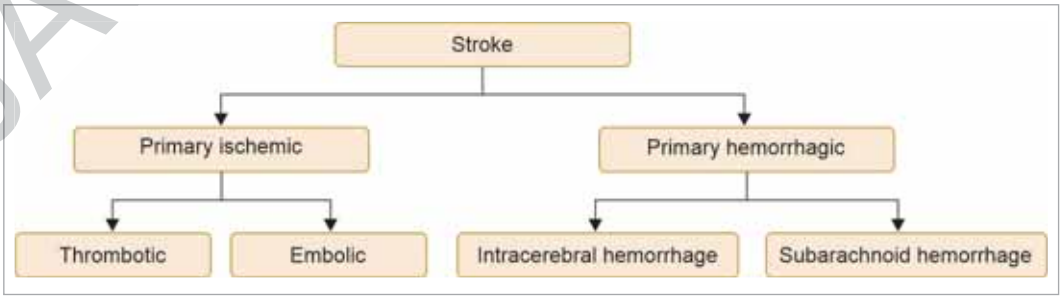
### Cortical Lesion

***Case Report***

- A 1-year-old girl was seen to fall backward after slipping on the floor. She cried immediately but did not lose consciousness.
- She then slept for some time.
- On waking up, she was noted not to be using her left hand.
- By the following day she was also not using her left leg.

**BOX 1: Symptoms of stroke.**

- Cortical/subcortical
- *Hemiplegic stroke*:
  - Hemiparesis, monoparesis, or quadriparesis
  - Monocular or binocular visual loss, visual field deficits, diplopia
  - Dysarthria, ataxia, vertigo
  - Aphasia, sudden ↓ in consciousness, neglect (nondominant)




**FLOWCHART 1:** Types of stroke in children.

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*Diagnosis*: Striatocapsular infarction after mild head trauma in children **(Figs. 1 and 2)**

*Possible mechanism*: Mechanical disruption to the perforating branches of middle cerebral artery (MCA), intimal trauma, and subsequent thrombosis, or arterial spasm induced by trauma. They can have associated mineralizing angiopathy as evidenced by calcification in the basal ganglia region **(Figs. 3A and B)**.

**TABLE 1: Arterial territory and stroke.**

| Vascular territory | Symptoms |
|---|---|
| Internal carotid artery | Hemiparesis, aphasia, and hemianopsia |
| Anterior cerebral artery | Hemiparesis, especially leg |
| Middle cerebral artery | Arm hemiparesis, hemianopsia, and aphasia |
| Posterior cerebral artery | Hemianopsia, ataxia, hemiparesis, and dizziness |
| Basilar artery | Breath, sensory or balance disturbances, ataxia, nystagmus, opisthotonus, tremor, and vomiting |
| Cerebellar artery | Sensory disturbances, headache, fever, vomit, and cerebellar signs |



**FIG. 1:** Left hemiplegia conjugate deviation of eye to right.

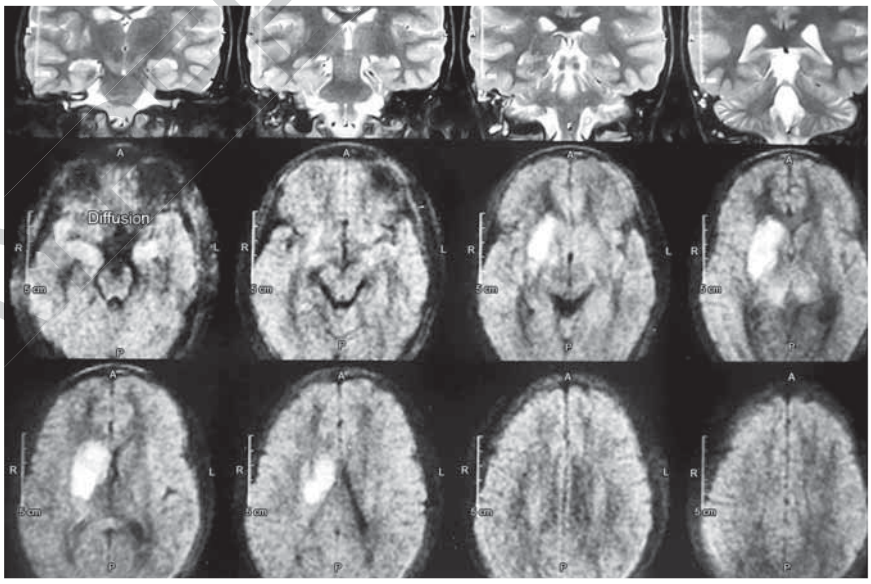
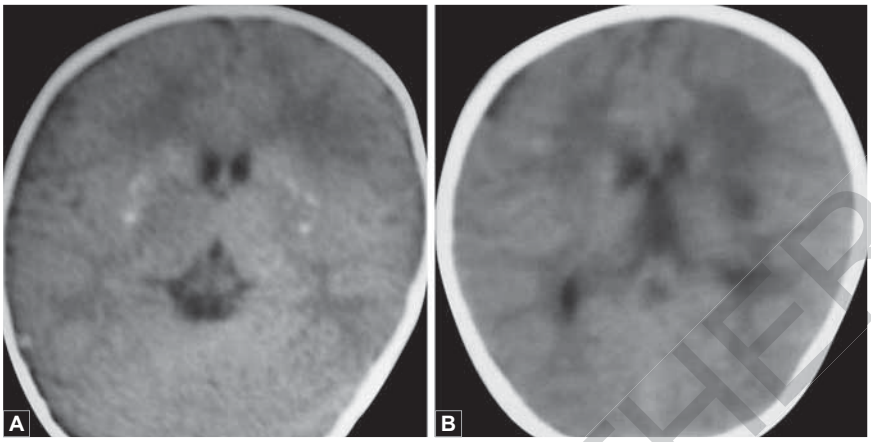



**FIG. 2:** Basal ganglia stroke due to mild head trauma in pediatric age diffusion-weighted imaging (DWI).

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**FIGS. 3A AND B:** Computed tomography (CT) scan at the time of admission showing no obvious parenchymal injury calcification at basal ganglia, and (B) report CT scan showing an area of hypodensity in left putamen suggestive of (s/o) ischemic parenchymal injury.

Arterial territory-involved strokes are described in **Table 1**.

- Brainstem and cerebellum
- *Crossed hemiplegic stroke*:
  - Gait or limb ataxia, vertigo or tinnitus, nausea, and vomiting
  - Hemiparesis or quadriparesis
  - Hemisensory loss or sensory loss of all four limbs
  - Eye movement abnormalities, diplopia, or nystagmus
  - Oropharyngeal weakness or dysphagia
  - Crossed signs (ipsilateral face and contralateral body)

## CAUSES OF ARTERIAL ISCHEMIC STROKE

Arterial ischemic stroke is the focal brain infarction that results from occlusion of the brain arteries or their branches.[4] Most etiologies for AIS are well established; some represent only potential associations. Though many are idiopathic most children have identifiable, multiple risk factors.[5] Three main categories are arteriopathic, cardiac, and hematological **(Fig. 4)**.

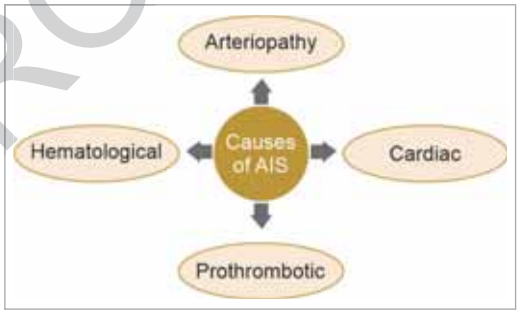



**FIG. 4:** Arterial ischemic stroke (AIS)—etiology.

*Different types of arteriopathy*: Arteriopathy makes up to >50% of the causes of AIS. They are:

- Focal cerebral arteriopathy (FCA)/transient cerebral arteriopathy, e.g., post-varicella angiopathy
- Childhood primary angiitis of the CNS (cPACNS)
- Systemic/secondary vasculitis
- Craniocervical arterial dissection (CCAD)
- Moyamoya disease
- Arterial infection (e.g., bacterial meningitis, tuberculosis)
- Fibromuscular dysplasia
- Vasospasm (e.g., Call–Fleming syndrome)

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- Congenital arterial abnormalities (e.g., PHACES syndrome)
- Idiopathic

*Imaging*: Transient cerebral arteriopathy

*Stenosis* distal part of internal carotid and initial segments and branches of anterior and/or middle cerebral artery—within 3 months; 6 months—nonprogression (or regression) of arterial lesions

FCA dissection type (FCA-d) = Intracranial arterial dissection of the anterior circulation, typically with trauma.

*Characteristics associated with FCA-inflammation type (FCA-i)*:

- Presentation with dysarthria
- Smaller infarcts in the lenticulostriate territory
- Infarct volume of <25 $cm^3$
- Arterial banding
- Isolated involvement of the M1 segment of the MCA **(Fig. 5)**

## Clinical Features of Post-varicella Arteriopathy

Varicella zoster virus (VZV) infection of cerebral arteries produces a vasculopathy, manifesting most often as ischemic stroke and less often as hemorrhagic stroke. Vasculopathy can occur after either primary infection with VZV (i.e., varicella; chickenpox) or after viral reactivation (i.e., herpes zoster; shingles—with immune compromised state). In children, it presents as a reversible transient type typically affecting lenticulostriate branches, which resolves over months **(Figs. 6 to 8)**.

## Craniocervical Arterial Dissection in Children (Figs. 9 to 11)

The International Pediatric Stroke Study defines CCAD as:

- Angiographic double lumen, intimal flap, or pseudo aneurysm, or, on axial T1 fat saturation magnetic resonance imaging (MRI) images, a "bright crescent sign" **(Fig. 11)** in the arterial wall

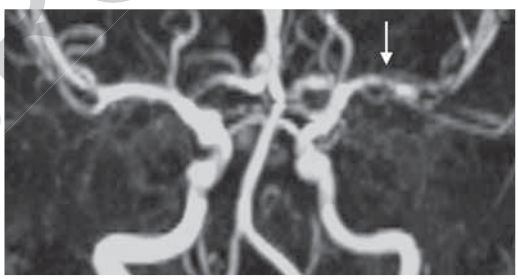
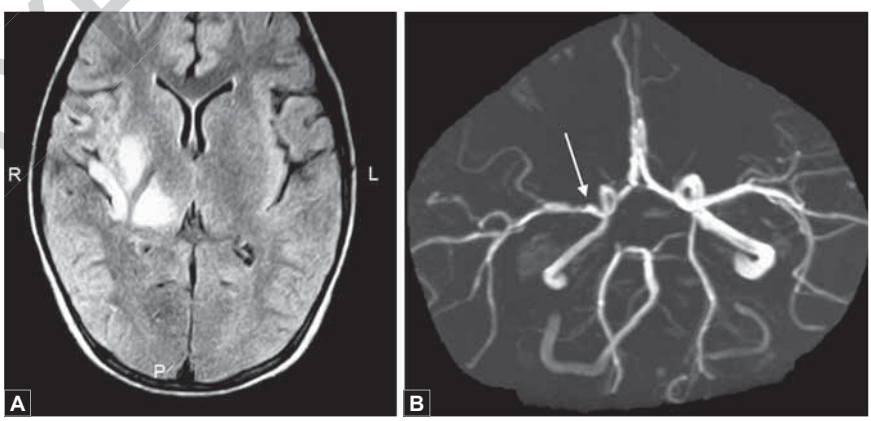


**FIG. 5:** Focal stenosis of the left middle cerebral artery (MCA) M1 segment.




**FIGS. 6A AND B:** Post-varicella arteriopathy. (A) Magnetic resonance imaging (MRI)—multifocal lesions involving (right) basal ganglia, thalamus; (B) Magnetic resonance angiography—middle cerebral artery narrowing, which disappeared after 3 months.

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**FIG. 7:** Herpes zoster left side of chest.

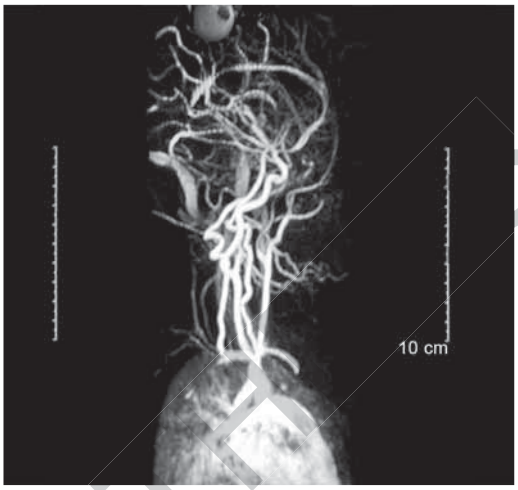



**FIG. 10:** Craniocervical arterial dissection in children—left internal carotid dissection.



**FIG. 8:** Left hemiplegia in a child with varicella zoster.



**FIG. 11:** "Bright crescent sign" in the left carotid arterial wall.

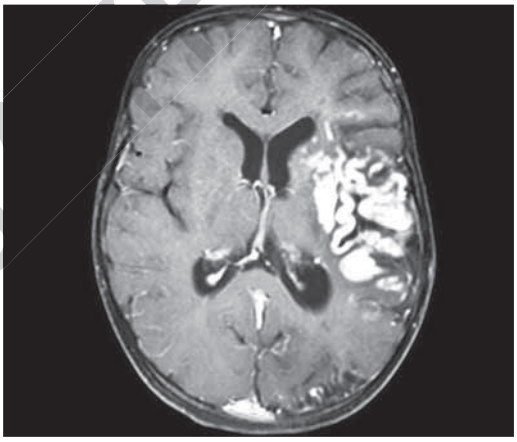


**FIG. 9:** Left middle cerebral artery (MCA) infarct.

- Cervical or cranial trauma, or neck pain, <6 weeks preceding angiographic findings of segmental arterial narrowing (or occlusion) located in the cervical arteries
- Angiographic segmental narrowing (or occlusion) of the vertebral artery at the level of the C2 vertebral body, even without known traumatic history [6]
- The most common location for carotid CCAD is 2–3 cm above the carotid bulb.

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- This is because of their mobility and proximity to bony projections of the cervical spine.
- The vertebral artery is particularly vulnerable at its tortuous course around the C1–C2 lateral masses and through the transverse foramina.
- *Genetic disorders associated with cervical and cerebral arteriopathy*:
  - Neurofibromatosis type 1
  - Alagille syndrome
  - Williams syndrome
  - PHACES syndrome
  - Trisomy 21

## MOYAMOYA DISEASE AND RADIOLOGICAL FEATURES

Moyamoya disease is a progressive, occlusive disease of the cerebral vasculature with particular involvement of the circle of Willis and the arteries that feed it with formation of abnormal networks of small collaterals. The term moyamoya (Japanese for "puff of smoke") refers to the appearance on angiography of abnormal vascular collateral networks that develop adjacent to the stenotic vessels. Cause is usually idiopathic but can be associated with sickle cell disease (SCD), Down syndrome, neurofibromatosis type I, Williams syndrome, postradiation vasculopathy, and congenital arteriopathy. Children may have hemiparesis, monoparesis, sensory impairment, involuntary movements, headaches, dizziness, or seizures. Mental retardation or persistent neurologic deficits may be present. These may be preceded by recurrent alternating transient ischemic attacks.

Cerebral angiography digital or magnetic resonance angiography (MRA) is the gold standard for the diagnosis. The following findings are seen:

- Stenosis or occlusion at the terminal portion of the internal carotid artery or the proximal portion of the anterior or middle cerebral arteries
- Abnormal vascular networks in the vicinity of the occlusive or stenotic areas
- Bilaterality of the described findings (although some patients may present with unilateral involvement and then progress)

## CENTRAL NERVOUS SYSTEM VASCULITIS

- Primary angiitis of the CNS and vasculitis associated with systemic illness (secondary)
- Primary angiitis of the CNS (PACNS) represents vasculitis confined to the CNS.
- Newly acquired neurological deficits or psychiatric symptoms in patients under 18 years
- With specific angiographic and/or histopathological features of CNS vasculitis
- *In the absence of an underlying systemic disorder or extra-CNS involvement*:
  - Large-vessel disease (angiography-positive) may be progressive (PcPACNS) or nonprogressive (NPcPACNS) and may manifest with focal deficits similar to an occlusive stroke.
  - Small-vessel disease (angiography-negative, biopsy-positive) results in language and cognitive problems.
    - Large-/medium-vessel cPACNS is diagnosed by angiography demonstrating features of vessel wall inflammation, wall swelling, and edema, and resulting luminal stenosis.
    - Monophasic, nonprogressive subtype (NPcPACNS)—limited to the proximal vessel segments of the anterior and/or middle cerebral artery and/or distal internal carotid artery of one hemisphere
    - Progressive subtype (PcPACNS)—affecting both proximal and distal vessel segments in one or both hemispheres

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*Childhood cerebral vasculitis (CCV) secondary to systemic vasculitis*: Identification of these vasculitis syndromes requires a comprehensive history and physical examination.[7] Vessel size-related vasculitis classification is given in **Figure 8**.

**Figure 12** describes vasculitis, with each one have stroke-like presentations.

- Coronavirus disease 2019 (COVID-19), including multisystem inflammatory syndrome in children (MIS-C), has been associated with a variety of cutaneous lesions, and vaso-occlusive disorders. Maternal COVID later presenting with neonatal stroke is reported.
- One systemic vasculitis with characteristic symptoms and sign is deficiency of adenosine deaminase 2 (DADA2)-related vasculitis.[8]
- DADA2 is a monogenic systemic vasculitis syndrome first described in 2014 characterized by inflammation of the medium- and small-sized blood vessels.
- Autosomal recessive disease associated with mutations of the *ADA2* gene (formerly known as *CECR1* gene).
- *p.Gly47Arg* mutation is the most common mutation identified among the first reported cases of DADA2 children who can present with brain stem or capsular stroke **(Figs. 13 to 19 and Tables 2 and 3)**
- Brainstem stroke

*Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL)* is caused by *NOTCH3* gene mutations. Manifestations include stroke, cognitive decline, and migraine.

*Cerebral autosomal recessive arteriopathy with subcortical infarcts and leukoencephalopathy (CARASIL)* is caused by *HTRA1* gene mutations. Manifestations include early-onset hair loss, spasticity, stroke, and personality changes.

A classification of immune-mediated inflammatory brain disorders are described in **Flowchart 2**.

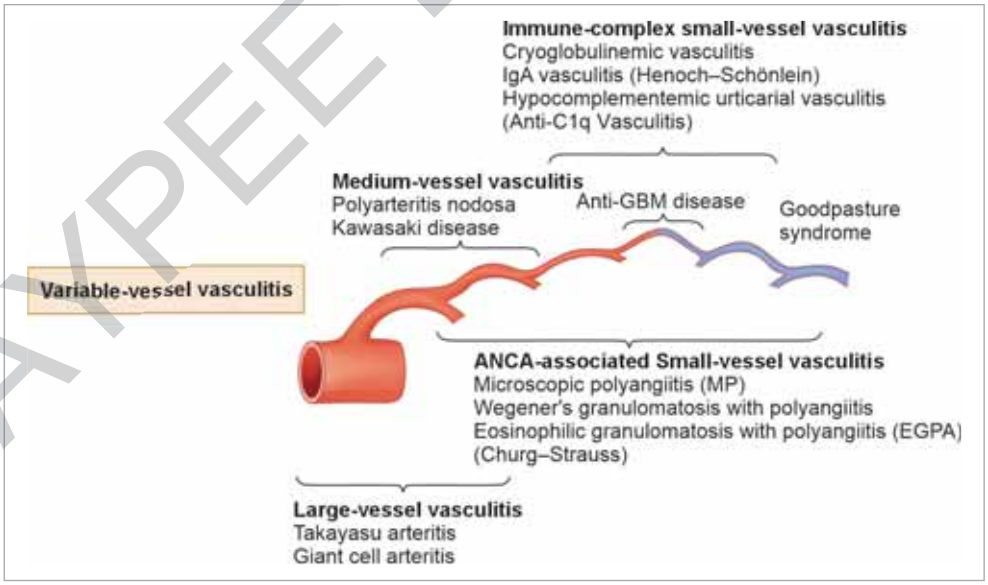
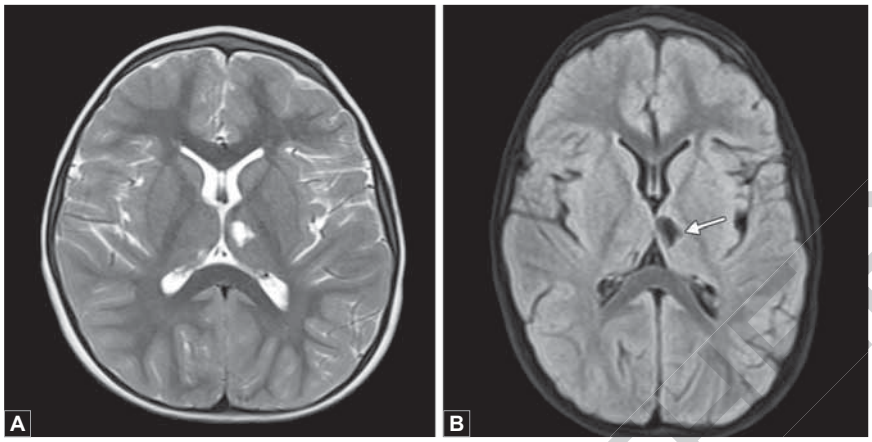



**FIG. 12:** Systemic vasculitis syndromes.

(ANCA: antineutrophil cytoplasmic antibody; GBM: glomerular basement membrane; IgA: immunoglobulin A)

*Source*: 2012 Chapel Hill Consensus Conference Nomenclature of Vasculitides.

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**FIGS. 13A AND B:** Deficiency of adenosine deaminase 2 (DADA2) vasculitis first stroke in thalamus.

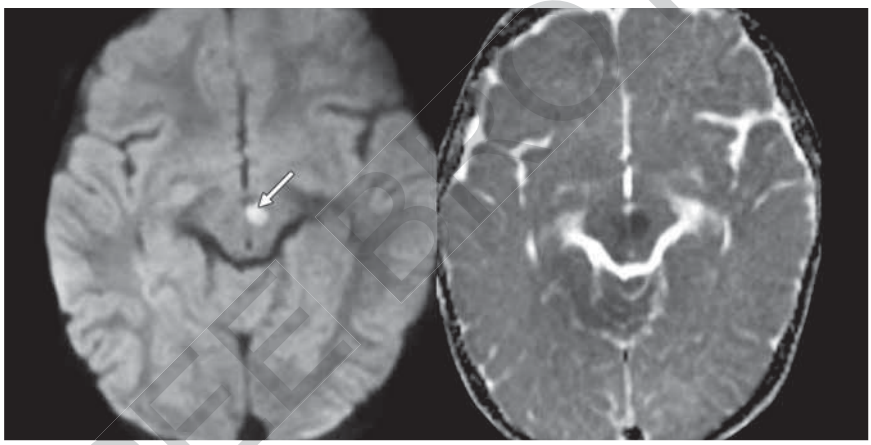


**FIG. 14:** Deficiency of adenosine deaminase 2 (DADA2); second stroke. Infract in the left ventral midbrain.



**FIG. 15:** Right facial, left third nerve palsy; acute-onset right ataxic hemiparesis.



**FIG. 16:** Deficiency of adenosine deaminase 2 (DADA2) erythema nodosum.

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**FIG. 17:** Deficiency of adenosine deaminase 2 (DADA2) hand vasculitis.



**FIG. 18:** Ankle joint swelling.

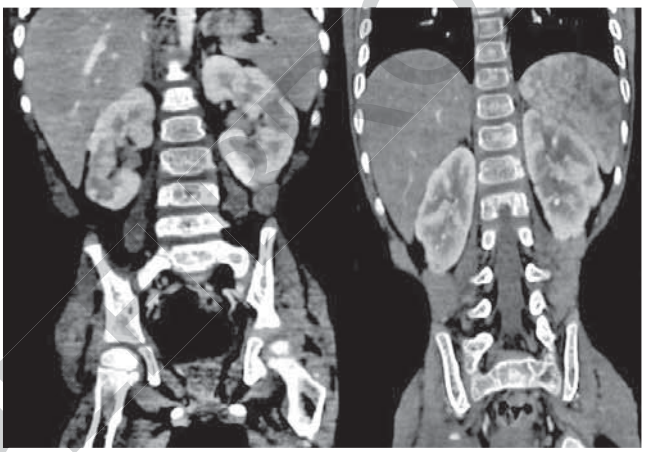


**FIG. 19:** Deficiency of adenosine deaminase 2 (DADA2) computed tomography (CT) angio kidney right renal scarring, microaneurysms with hypertension.

**TABLE 2: Deficiency of adenosine deaminase 2 (DADA2) enzyme deficiency.**

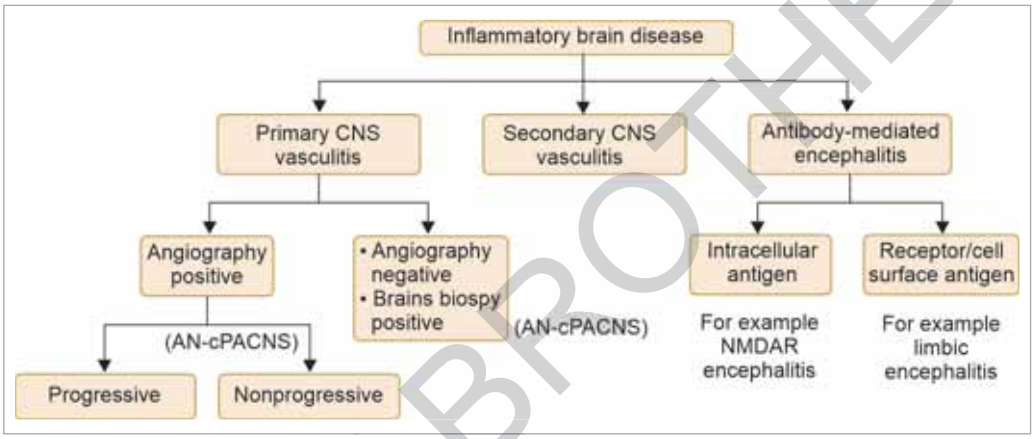
| Name | Date of sample | Plasma spot ADA2 activity mU/g protein |
|---|---|---|
| Vasudev Sminu | 06-17-2021 | 20.9 |
| Control | 06-17-2021 | 195.6 |
| *Previous dried plasma spots tested for ADA2 deficiency* | | |
| **Source of samples** | **ADA2 activity mU/g protein** | |
| ADA2 deficient ($n$ = 78) | 4.7 ± 4.8 (0–23.3) | |
| ADA2 carriers ($n$ = 41) at risk | 55.3 ± 22.0 (27.2–108.7) | |
| Not deficient ($n$ = 278) | 233.7 ± 176.8 (26.6–1186) | |
| Controls ($n$ = 106) | 130.0 ± 53.2 (24.9–285) | |

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**TABLE 3: *DADA2* gene mutation.**

| Result: Following variant/s has been identified by Sanger Sequencing | | | | | | |
|---|---|---|---|---|---|---|
| **Gene** | **Location** | **Variant/ Chr:bp** | **Nucleotide position/ change** | **Amino acid position/ change** | **Allele zygosity** | **Clinical significance** |
| *ADA2* | Exon 2 | SNV/22: 17209539 | ENST00000399837.8:c. I39G>A | p.Gly47Arg | Homozygous | Pathogenic |

*Note*: Patient's genomic DNA contains a known variant in the gene *ADA2* (exon 2), which has been previously reported in association with polyarteritis nodosa (childhood onset).




**FLOWCHART 2:** Immune-mediated brain disorders.

(AN-cPACNS: angiography-negative childhood primary angiitis of the central nervous system; AP-cPACNS: angiography-positive cPACNS; NMDAR: N-methyl-D-aspartate receptor)

## PRESENTATIONS OF CEREBRAL SINOVENOUS THROMBOSIS

Thrombosis in the CNS is called as cerebral sinovenous thrombosis. Cerebral venous sinus (sinovenous) thrombosis (CSVT) is an increasingly recognized cause of childhood and neonatal stroke. One in four cases of childhood ischemic stroke is CSVT. The incidence of childhood CSVT varies between 0.4 and 0.7 per 100,000 children per year. More than 40% of childhood CSVT occurs within the neonatal period. Risk factors for CSVT include systemic causes like fever, infection, dehydration, congenital heart disease, nephrotic syndrome, systemic lupus erythematosus, prothrombotic disorders, and malignancy. CSVT can propagate in response to local venous stasis in relation to head trauma, CNS tumors, recent intracranial surgery, otitis media, and mastoiditis.

*Clinical presentation*: The clinical manifestations of CSVT are nonspecific, may be subtle **(Fig. 20)**, and may overlap with predisposing conditions such as infection and dehydration. Seizures, altered levels of consciousness and encephalopathy, focal neurologic deficits (cranial nerve palsies, hemiparesis, hemisensory loss), and diffuse neurologic symptoms (headache, nausea, emesis) may result.

Also CSVT should be suspected with pseudotumor cerebri-like syndrome, hydrocephalus, subdural hemorrhage (SDH), subarachnoid hemorrhage (SAH),

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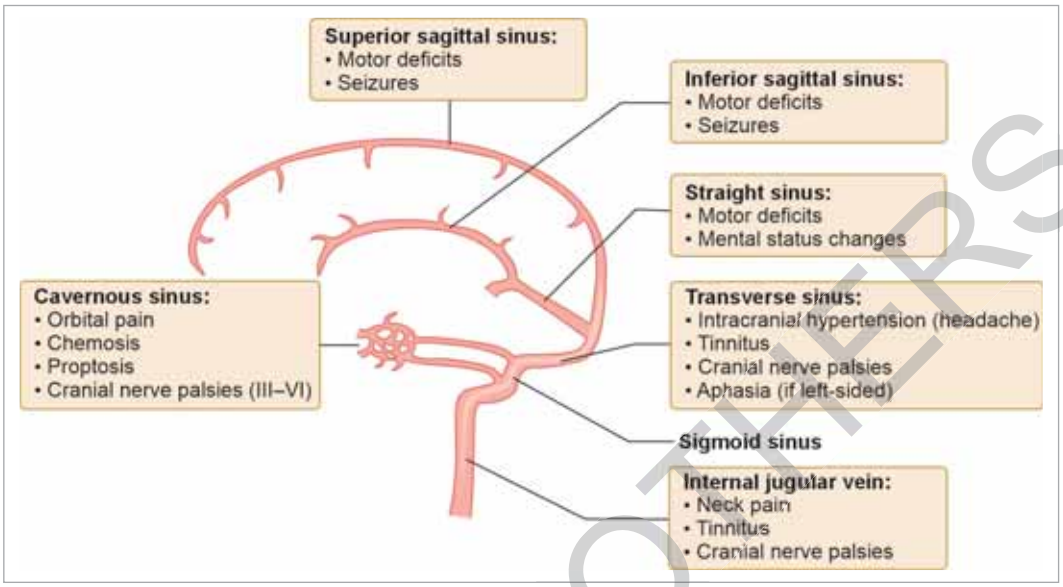



**FIG. 20:** Clinical feature of cerebral venous thrombosis.

intracerebral hemorrhage (ICH), and infarction not involving a specified vascular territory or bilateral.

## EVALUATION OF STROKE IN A CHILD

In approaching a child with stroke, history and physical examination must try to find out any associated cardiac disease, SCD, infections, dehydration, chicken pox, rheumatic fever, trauma, malignancy, thrombotic events, or family history of thrombotic events and hypertension.

*Investigations must be tailored*:

- To confirm the diagnosis/differential diagnosis
- To identify the predisposing factors

*To confirm the diagnosis*:

- MRI brain with contrast or computed tomography (CT) scan if MRI not possible
  - Can differentiate infarcts and hemorrhage from demyelination, tumors, and encephalitis
- Cerebrospinal fluid (CSF) study in suspected intracranial infections
- Electroencephalogram (EEG)
- SERUM/CSF lactate—if MELAS (Mitochondrial Encephalopathy, Lactic Acidosis, and Stroke-like episodes) is suspected

*To identify the predisposing factors*:

- Hemoglobin and peripheral smear for sickle cell anemia, Iron deficiency anemia is found to be associated with increased stroke incidence
- Prothrombin time (PT), activated partial thromboplastin time (APTT)—in hemorrhagic stroke
- X-ray chest, electrocardiogram (ECG), ECHO
- Erythrocyte sedimentation rate (ESR), antinuclear antibody (ANA), double-stranded DNA antibody (dSDNA)
- Contrast MRA of intracranial and neck vessels in acute ischemic stroke and arteriovenous malformation (AVM)
- MR venogram (MRV) in venous sinus thrombosis

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- *Profile of prothrombotic factors*:
  - Protein C
  - Protein S
  - Factor 5 Leiden
- Carotid Doppler
- Digital subtraction angiography—if MRA negative and dissection/aneurysm is suspected (especially in posterior circulation stroke)

## MANAGEMENT OF A CASE OF STROKE IN A CHILD

Following is the management protocol as per American Heart Association (AHA) guidelines 2008.

- Supportive measures for AIS should include:
  - Control of fever, maintenance of normal oxygenation, control of systemic hypertension, and normalization of serum glucose levels (Class I, Level of Evidence C)
  - It is reasonable to treat dehydration and anemia in children with stroke.
  - Prophylactic administration of antiepileptic medications in children with ischemic stroke is not necessary (Class III, Level of Evidence C).
- *Specific treatment*:
  - *SCD*: Acute management of ischemic stroke due to SCD should include optimal hydration, correction of hypoxemia, and correction of systemic hypotension (Class I, Level of Evidence C). Periodic transfusions to reduce the percentage of sickle hemoglobin are effective for reducing the risk of stroke in children 2–16 years of age with abnormal transcranial Doppler (TCD) results due to SCD and are recommended (Class I, Level of Evidence A).[5]
  - *Cervicocephalic arterial dissection*: Low-molecular-weight heparin (LMWH) followed by oral anticoagulation for 3–6 months
  - *Congenital heart disease producing embolic stroke*:
    - LMWH followed by oral anticoagulation for at least a year
    - Definitive surgical management of heart disease other than patent foramen ovale (PFO)
    - Resection of myxoma
    - Aspirin for PFO alone
  - Anticoagulation with LMWH is useful for long-term anticoagulation of children with a substantial risk of recurrent cardiac embolism, CSVT, dissection, and selected hypercoagulable states (Class I, Level of Evidence C).
  - The administration of LMWH or unfractionated heparin (UFH) may be considered in children for up to 1 week after an ischemic stroke pending further evaluation to determine the stroke's etiology (Class IIb, Level of Evidence).
  - Anticoagulation with warfarin is reasonable for the long-term anticoagulation of children with a substantial risk of recurrent cardiac embolism, CCAD, CVST, or selected hypercoagulable states (Class IIa, Level of Evidence C).
  - Aspirin is a reasonable option for the secondary prevention of AIS in children whose infarction is not due to SCD and in children who are not known to have a high risk of recurrent embolism or a severe hypercoagulable disorder (Class IIa, Level of Evidence C).
  - *Thrombolytic therapy for childhood stroke*:
    - Thrombolytic therapy with tissue plasminogen activator (tPA) may be considered in selected children with CVST (Class IIb, Level of Evidence C).
    - Until there are additional published safety and efficacy data,

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tPA is not recommended for AIS outside of a clinical trial (Class III, Level of Evidence C).

- *Modifying stroke risk factors in children*:
  - To treat iron deficiency because it may increase the risk of AIS in conjunction with other risk factors (Class IIa, Level of Evidence C)
  - Dietary improvement, the benefits of exercise, and the avoidance of tobacco products
- *Evaluation and treatment of hemorrhage in children*:
  - Children with a severe coagulation factor deficiency should receive appropriate factor replacement therapy, and children with less severe factor deficiency should receive factor replacement following trauma (Class I, Level of Evidence A).
  - Congenital vascular anomalies should be identified and corrected whenever it is clinically feasible. Similarly, other treatable hemorrhage risk factors should be corrected (Class I, Level of Evidence C).
- *Treatment of CSVT*:
  - *Class I Recommendations*:
    - Appropriate hydration
    - Control of epileptic seizures
    - Management of raised intracranial pressure (ICP)
    - Systemic antibiotics if bacterial infection is suspected
    - Periodic assessment of visual field and visual acuity
  - *Class 2 Recommendations*:
    - Intravenous or subcutaneous heparin/LMWH followed by warfarin for 3–6 months even if there is hemorrhage
    - Repeat MRV to assess recanalisation
    - Investigate for thrombophilia

## GRAPHIC ARTERIAL STROKE IN CHILDREN

Graphic arterial stroke representation through pictures in children are due to cardiac causes **(Fig. 21)**, thrombotic causes **(Fig. 22)** including blood-formed elements, plasma, and metabolic causes.

## PEDIATRIC STROKE REHABILITATION

The recovery of each child is unique and requires a neurologist, physical medicine and rehabilitation (PM&R) physician, occupational therapist, physical therapist,

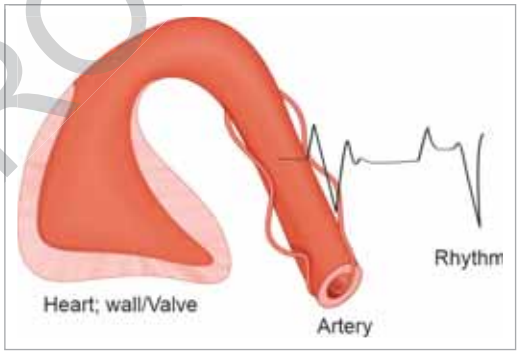



**FIG. 21:** Cardiac-related stroke causes.

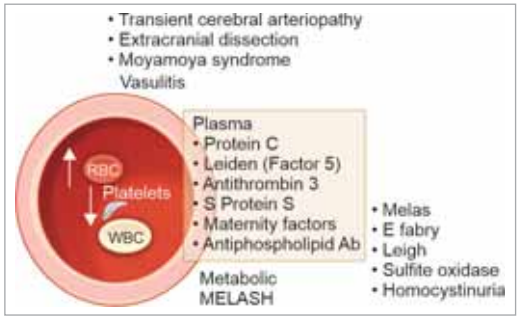



**FIG. 22:** Thrombotic etiology.

(MELASH: mnemonic for remembering metabolic stroke; RBC: red blood cell; WBC: white blood cell)

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speech therapist, neuropsychologist, social worker, school service officials, and nutritionist to develop an individual plan.[9]

- Generally, includes physiotherapy, occupational therapy (OT), speech therapy to help children to improve motor function, speech and behavioral issues; OT will help to achieve independence with daily living skills [activity of daily living (ADL)] when facing the effects of stroke. OT also helps to restore upper extremity functioning after stroke.
- Long-term effects may include comorbidities associated with cerebral palsy (CP):
  - Intellectual disability—20%
  - Seizures—30%
  - Behavior problems 30–50%
  - Speech and language—40%
  - Hearing—8–18%
  - Eye problem—30%

  So the specific rehabilitative measures to restore these functions also to be undertaken. Though aphasia is uncommon in pediatric stroke, dysarthria, phonation difficulty, impaired speech intelligibility are the common language problems. They occur due to spasticity of muscles of tongue, mouth, and larynx, and corticobulbar dysfunction as pediatric stroke may have bilateral involvement.
- Gross motor functional classification system (GMFCS) can be used to assess the functional status of ambulation.
  - *GMFCS*:

    *Level 1*: Clumsy child; no assistive devices

    *Level 2*: Walks independently but limited in outdoor activities

    *Level 3*: Walks with assistive mobility devices

    *Level 4*: Self-mobility severely limited even with assistive devices

    *Level 5*: No self-mobility even with assistive devices
- Hand function can be assessed by manual ability classification system.
  - *Manual ability classification system*:

    *Level 1*: Handles object easily and successfully

    *Level 2*: Handles most objects, but with a reduced quality and/or speed of achievement

    *Level 3*: Handles objects with difficulty and needs help to prepare and/or modify activities

    *Level 4*: Handles a limited selection of easily managed objects in adapted situations

    *Level 5*: Does not handle objects
- *Assessment of spasticity*:
  - Modified Ashworth scale:
    - *Early intervention measures for established stroke*: In early intervention programs, the primary caregiver (either a pediatrician or a developmental therapist) works to create a developmental setting in which the key to child's improvement is by mother–child participation.

    The basis of early intervention is that there are critical periods of development and if interventions happens before that, delay and disabilities can be minimized. This focuses on mobility, manipulation, and communication.

    So anticipate neonatal stroke in high-risk babies including preterm and use early screening for motor milestone delay and tone change by Amiel–Tison angles.

    Regular passive exercises can be done at home by the parents as per the assessment.

    By utilizing the head steadiness, sitting, standing and walking developmental observation, and manipulative techniques can be utilized

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