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Heterogeneous Clinical Spectrum of Moyamoya Disease in a Tertiary Care Centre: A Seven-Patient Case Series Highlighting Diagnostic Challenges

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ABSTRACT

Background and Objectives: Moyamoya disease (MMD) is a rare, progressive cerebrovascular disorder characterised by bilateral stenosis or occlusion of the internal carotid arteries and their branches, with compensatory formation of a hazy network of collateral vessels. The disease manifests with diverse clinical presentations—including ischaemic stroke, haemorrhage, headache, and seizures—making early diagnosis challenging, particularly in resource-limited settings. This case series aims to describe the heterogeneous clinical, radiological, and angiographic spectrum of MMD encountered at a tertiary neurology centre in India, and to highlight the diagnostic and therapeutic challenges therein.

Methods: We retrospectively reviewed records of seven patients diagnosed with MMD at our tertiary care centre. Data were collected on demographics, clinical presentation, neuroimaging (MRI brain and MR/CT angiography), digital subtraction angiography (DSA) grading per Suzuki staging, management strategies, additional autoimmune and haematological workup, laterality, and functional outcome measured by the modified Rankin Scale (mRS) at the time of last follow-up.

Results: The cohort comprised six females and one male (age range: 7–39 years; median age 20 years). Presentations included ischaemic stroke (n=2), intracranial haemorrhage (n=1), headache (n=3), and epilepsy (n=2). One patient had concurrent features of neurofibromatosis type 1 (NF1), representing a syndromic form of moyamoya. DSA grading ranged from Stage 3 to Stage 4, with bilateral involvement in all cases. Serum homocysteine was elevated in one patient. Functional outcomes were generally favourable, with five of seven patients achieving mRS ≤2 at follow-up.

Conclusions: MMD in the Indian context presents with significant clinical heterogeneity, affecting predominantly young females. Syndromic associations such as NF1 should be actively sought. Autoimmune and haematological workup is essential to exclude secondary moyamoya syndrome. Expeditious diagnosis and individualised management can yield acceptable functional outcomes even without surgical revascularisation in selected cases.

Keywords*: Moyamoya disease; cerebrovascular disease; intracranial stenosis; collateral circulation; Suzuki staging; neurofibromatosis; stroke in the young; case series.*

INTRODUCTION

Moyamoya disease (MMD) is a chronic, idiopathic, occlusive cerebrovascular disorder first described by Takeuchi and Shimizu in 1957 and formally named by Suzuki and Takaku in 1969, the term "moyamoya" deriving from the Japanese word for "puff of smoke," describing the characteristic angiographic appearance of basal collateral vessels.[1,2] The disease is characterised by progressive stenosis or occlusion of the terminal internal carotid arteries (ICA) and the proximal portions of the anterior (ACA) and middle cerebral arteries (MCA), with a compensatory network of fine collateral vessels developing at the base of the brain.[3]

The global prevalence of MMD is estimated at approximately 6 per 100,000 population, with the highest burden reported in East Asian countries, particularly Japan and South Korea.4 Indian data remain sparse, though emerging case series suggest that the disease is not uncommon in the South Asian context and may carry distinct phenotypic features.[5,6] MMD exhibits a bimodal age distribution with peaks in childhood (presenting predominantly with ischaemia) and adulthood (presenting with haemorrhage), and a female predilection (female-to-male ratio approximately 1.8:1).[4]

The aetiology of MMD remains poorly understood. Genetic predisposition involving the RNF213 gene (particularly the founder variant p.R4810K, common in East Asian populations) is well-established, though Indian-specific variants remain under characterisation.[7] Secondary moyamoya syndrome—a morphologically identical entity occurring in the setting of predisposing conditions such as neurofibromatosis type 1 (NF1), sickle cell disease, Down syndrome, and autoimmune vasculitis—must be excluded before a diagnosis of idiopathic MMD is rendered.[8]

Diagnosis rests on magnetic resonance imaging and angiography, supplemented by conventional DSA for definitive grading. The Suzuki–Takaku staging system (Stage 1–6) quantifies the degree of arterial occlusion and collateral formation and carries prognostic and therapeutic implications.[9] Management strategies include antiplatelet therapy, neuroprotection, and surgical revascularisation—both direct (extracranial–intracranial bypass) and indirect (encephaloduroarteriosynangiosis, EDAS)—with the latter reserved for progressive or surgically amenable disease.[10]

Despite growing recognition, diagnostic delays remain common in India due to limited awareness, phenotypic overlap with other vasculopathies, and restricted access to advanced neuroimaging and DSA facilities. Herein, we report a case series of seven patients with MMD diagnosed at a single tertiary neurology centre, highlighting the heterogeneity of clinical presentations, the importance of a systematic diagnostic workup, and the challenges in management particular to the Indian setting.

METHODS

This is a retrospective, observational case series. Records of all patients diagnosed with MMD at the Neurology Department of our tertiary care centre over a five-year period were reviewed. The diagnosis of MMD was established based on the modified Research Committee on Moyamoya Disease criteria: (i) stenosis or occlusion at the terminal ICA and/or proximal ACA/MCA on cerebral angiography (MRA/CTA/DSA); (ii) presence of abnormal vascular networks in the vicinity of the occlusion on the arterial phase; (iii) bilateral involvement; and (iv) exclusion of secondary causes (atherosclerosis, autoimmune arteritis, intracranial tumour, irradiation, and Down syndrome).[9]

Data collected included: patient demographics (age, sex), mode of clinical presentation, neuroimaging findings (MRI brain sequences and vascular imaging), DSA Suzuki–Takaku stage, laterality, medical management, additional workup (autoimmune panel—antinuclear antibody [ANA], extractable nuclear antigen [ENA], anti-neutrophil cytoplasmic antibody [ANCA]; haematological workup—complete blood count [CBC], peripheral blood film [PBF], haemoglobin electrophoresis; metabolic workup—serum homocysteine; and vascular workup—renal Doppler/angiography where indicated), and functional outcome assessed by the modified Rankin Scale (mRS) at the most recent follow-up. Informed consent was obtained from all patients or their legal guardians for publication of clinical details.

Cases with unilateral involvement only or with a confirmed secondary aetiology attributable to radiation, atherosclerosis, or other definite underlying systemic disease were excluded. This study was approved by the Institutional Ethics Committee.

RESULTS

The cohort comprised seven patients (six females, one male) with a median age of 20 years (range 7–39 years). All cases demonstrated bilateral involvement. DSA Suzuki staging ranged from Stage 3 (n=3) to Stage 4 (n=4). Five of seven patients (71.4%) achieved mRS ≤2 at follow-up. A comprehensive summary is presented in Table 1.

Table 1: Summary of Clinical, Radiological, and Outcome Data in Seven Patients with Moyamoya Disease

Patient	Age/Sex	Presentation	Imaging Findings	Suzuki Stage	Management	Laterality	mRS	Additional Workup
Anisha	15/F	Headache, vomiting, fever, bilateral visual blurring (acute	Bilateral ICA–ACA–MCA–PCA occlusion + collaterals; cortical right ICA aneurysm	Stage 4	Aspirin 75 mg OD; Atorvastatin 20 mg HS (surgery declined)	Bilateral	1	ANA –ve; ENA –ve; HbEP –ve

Patient	Age/Sex	Presentation	Imaging Findings	Suzuki Stage	Management	Laterality	mRS	Additional Workup
		onset)						
Chetana Chhipa	20/F	Migraine headache ×10y; GTCS ×10y; subacute slurring of speech; urinary incontinence	Subacute right frontotemporal infarcts; bilateral supraclinoid ICA/ACA/MCA non-opacification + collaterals; right PCA attenuation	Stage 3	Aspirin 150 mg; Atorvastatin 40 mg; management of hyperhomocysteine	Bilateral	1	Homocysteine: RAISED; ANA –ve; ENA –ve; HbEP –ve
Farida Bano	39/F	Daily holocranial pulsatile headache ×1y; photophobia; remote left UL weakness (25y prior)	Diffuse bilateral ICA/ACA narrowing; multiple collaterals	Stage 3	Cilostazol; Atorvastatin; Amitriptyline	Bilateral	2	Renal Doppler/Angio –ve; ANA, ENA, ANCA –ve; HbEP –ve
Laxmi	7/F	Recurrent focal seizures with impaired awareness ±GTCS ×5y	Right frontoparietal encephalomalacia/gliosis; left hippocampal sclerosis; bilateral supraclinoid ICA narrowing + collaterals	Stage 4 (R>L)	Aspirin 75 mg; Atorvastatin 20 mg; Levetiracetam; Clobazam; Sodium Valproate	Bilateral	1	Negative
Narender	34/M	Sudden-onset severe headache during exertion; visual blurring; vomiting ×4d	Right subthalamic/hypothalamic haemorrhage + IVH; bilateral supraclinoid ICA/MCA/ACA narrowing; interpeduncular collaterals	Stage 4	Atorvastatin 40 mg; Nimodipine; Antioedema measures	Bilateral	2	Negative
Rekha	12/F	Acute ischaemic stroke: left hemiparesis + UMN facial palsy; prior similar episode 1y back	Diffuse bilateral intracranial ICA narrowing + collaterals	Stage 3	Aspirin; Atorvastatin; Nimodipine	Bilateral	3	PBF: microcytic hyperchromic anaemia + target cells; HbEP sent; ANA, ANCA, Anti-CCP pending
Sahiba Bano	26/F	Headache ×2y; syncope with vertigo	Severe bilateral cavernous/supraclinoid ICA stenosis; absent bilateral ACA/MCA	Stage 4	Aspirin; Atorvastatin	Bilateral	1	CBC: anaemia; HbEP/sickle cell –ve;

Patient	Age/Sex	Presentation	Imaging Findings	Suzuki Stage	Management	Laterality	mRS	Additional Workup
		×3–4 episodes; NF1 stigmata on examination	flow; extensive pial/parenchymal micro-collaterals; dominant PCA					ANA/ENA – ve

Abbreviations: ICA = internal carotid artery; ACA = anterior cerebral artery; MCA = middle cerebral artery; PCA = posterior cerebral artery; GTCS = generalised tonic–clonic seizures; UMN = upper motor neuron; UL = upper limb; NF1 = neurofibromatosis type 1; IVH = intraventricular haemorrhage; mRS = modified Rankin Scale; PBF = peripheral blood film; HbEP = haemoglobin electrophoresis; ANA = anti-nuclear antibody; ENA = extractable nuclear antigen; ANCA = anti-neutrophil cytoplasmic antibody; OD = once daily; HS = at bedtime; R > L = right greater than left.

Individual Case Descriptions

Case 1: Anisha, 15-Year-Old Female

A 15-year-old girl presented with a 5-day history of acute-onset holocranial headache (worse in the recumbent position), vomiting, fever, and bilateral blurring of vision of mild-to-moderate severity. There was no history of seizure, loss of consciousness, or focal neurological deficit. Neurological examination was unremarkable apart from mild bilateral papilloedema. MRI brain with MR angiography revealed extensive vaso-occlusive disease involving bilateral ICA–ACA–MCA–PCA with prominent collateral formation consistent with moyamoya disease, and an incidental aneurysm arising from the cortical branch of the right ICA. DSA confirmed Stage 4 bilaterally (Suzuki–Takaku). Autoimmune workup (ANA, ENA) and haemoglobin electrophoresis were negative. The patient and family declined surgical revascularisation; she was commenced on aspirin 75 mg once daily and atorvastatin 20 mg at bedtime. At follow-up, she achieved a modified Rankin Scale (mRS) score of 1.

Case 2: Chetana Chhipa, 20-Year-Old Female

A 20-year-old woman presented with a decade-long history of migraine-type headache and generalised tonic–clonic seizures (GTCS), followed by acute worsening with fever (1.5 days), slurring of speech (4–5 days), and urinary incontinence (3–4 episodes). MRI brain demonstrated multiple T2/FLAIR hyperintensities with diffusion restriction in the cortical regions of the right frontal and right temporal lobes (subacute infarcts), and CSF-attenuated areas with surrounding FLAIR signal in the right parietal and temporal cortex consistent with gliosis from prior infarction. MR angiography showed non-opacification of the distal bilateral supraclinoid ICA and proximal ACA and MCA segments, with collateral networks in the bilateral capsuloganglionic region. The right PCA was attenuated; the left PCA was prominent with posterior collaterals. Findings were consistent with moyamoya disease, Suzuki Stage 3. Notably, serum homocysteine was elevated; ANA, ENA, and haemoglobin electrophoresis were negative. She was managed with aspirin 150 mg daily, atorvastatin 40 mg, and homocysteine supplementation. Outcome: mRS 1.

Case 3: Farida Bano, 39-Year-Old Female

A 39-year-old woman presented with a 1-year history of daily, holocranial, pulsatile headache with photophobia and severe intensity, relieved only partially on analgesic medication and altering sleep. There was a remote history of left upper limb weakness 25 years prior, for which she had received treatment and achieved partial recovery. There was no history of seizure, focal neurological deficit, dysphagia, or bowel/bladder disturbance. Clinical examination revealed no definitive focal deficits. MRI angiography demonstrated diffuse narrowing of bilateral ICA and ACA with multiple collaterals, graded Stage 3 on Suzuki staging. An extensive secondary workup including renal Doppler and angiography, ANA, ENA, ANCA, and haemoglobin electrophoresis was entirely negative. She was managed with cilostazol, atorvastatin, and amitriptyline for headache prophylaxis, achieving mRS 2 at follow-up.

Case 4: Laxmi, 7-Year-Old Female

A 7-year-old girl was referred for recurrent focal seizures with impaired awareness, occasionally evolving to bilateral tonic activity, persisting over 5 years. MRI brain revealed a large area of encephalomalacia and gliosis involving the right frontoparietal region and basal ganglia with ex-vacuo dilatation of the right lateral ventricle, consistent with a remote ischaemic insult. Additionally, left hippocampal volume loss with FLAIR hyperintensity was identified, indicative of hippocampal sclerosis secondary to long-standing unilateral hemispheric injury. Cerebral angiography confirmed bilateral narrowing of the supraclinoid ICA with collateral formation, consistent with moyamoya disease (Stage 4, right > left). Secondary autoimmune and haematological workup was negative. Management comprised aspirin 75 mg daily, atorvastatin 20 mg at bedtime, and anti-epileptic therapy (levetiracetam, clobazam, sodium valproate). She maintained mRS 1 at follow-up.

Case 5: Narender, 34-Year-Old Male

A 34-year-old male presented with sudden-onset severe headache during vigorous exercise (weight lifting), accompanied by blurring of vision and multiple episodes of vomiting, over 4 days. This was the only male patient in the cohort. CT brain demonstrated acute haemorrhage in the right subthalamic and hypothalamic region with intraventricular extension into the bilateral lateral, third, and fourth ventricles; no evidence of midline shift or hydrocephalus. CT angiography revealed diffusely narrow calibre of bilateral cavernous and supraclinoid ICA segments, bilateral proximal MCA and ACA narrowing, and multiple tortuous collaterals in the interpeduncular and perimesencephalic cisterns, without aneurysm or atherosclerotic plaque, graded Stage 4. Secondary workup was negative. He was managed conservatively with atorvastatin 40 mg, nimodipine, and antioedema measures. Functional outcome: mRS 2.

Case 6: Rekha, 12-Year-Old Female

A 12-year-old girl presented with acute ischaemic stroke manifest as left hemiparesis with upper motor neuron (UMN) facial palsy. She had a documented history of a similar episode 1 year prior. MRI angiography demonstrated diffuse narrowing of the intracranial ICA bilaterally with collateral formation, Suzuki Stage 3. Peripheral blood film revealed microcytic hyperchromic anaemia with target cells; haemoglobin electrophoresis was sent to exclude sickle cell disease. Abdominal ultrasound demonstrated a normal spleen. Autoimmune workup (ANA, ANCA, RA factor, Anti-CCP) was initiated; CT angiography of the renal and peripheral vessels was planned to exclude systemic vasculopathy. Medical management comprised aspirin, atorvastatin, and nimodipine. She had the least favourable outcome in the cohort at mRS 3, reflecting the severity of bilateral ischaemic injury.

Case 7: Sahiba Bano, 26-Year-Old Female — Syndromic Moyamoya (NF1)

A 26-year-old woman presented with a 2-year history of headache and 3–4 episodes of syncope with vertigo and loss of consciousness. On examination, she displayed stigmata of neurofibromatosis type 1 (NF1): cutaneous neurofibromas, multiple café-au-lait macules, scoliosis, Lisch nodules, and axillary/inguinal freckling. MRI brain with gadolinium and MR angiography demonstrated mild diffuse reduction in calibre of the bilateral petrous ICA, with severe stenosis of bilateral cavernous and supraclinoid ICA segments. Bilateral ACA and MCA flow signals were absent. The basilar artery and bilateral vertebral arteries were prominent, and the bilateral PCA were dominant with extensive pial and parenchymal micro-collaterals in bilateral cerebral deep nuclei, periventricular regions, and the quadrigeminal plate—consistent with a moyamoya angiographic pattern (Stage 4). No intracranial aneurysm was identified. Haematological workup including haemoglobin electrophoresis (thalassaemia/sickle cell screen) and ANA/ENA panel were negative. She was managed with aspirin and atorvastatin, achieving mRS 1.

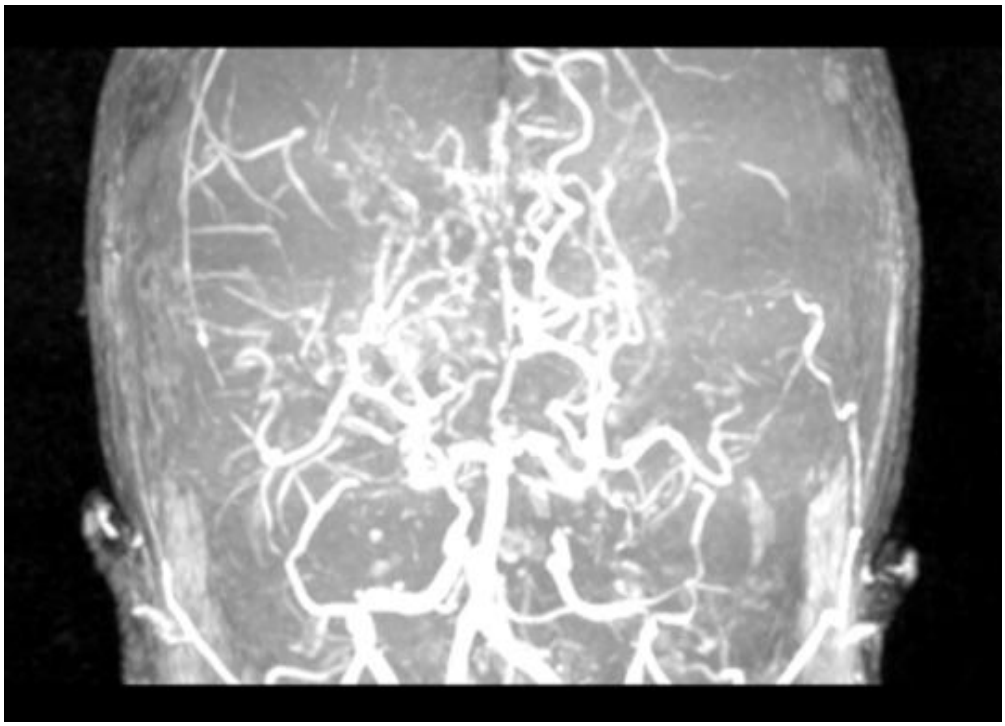
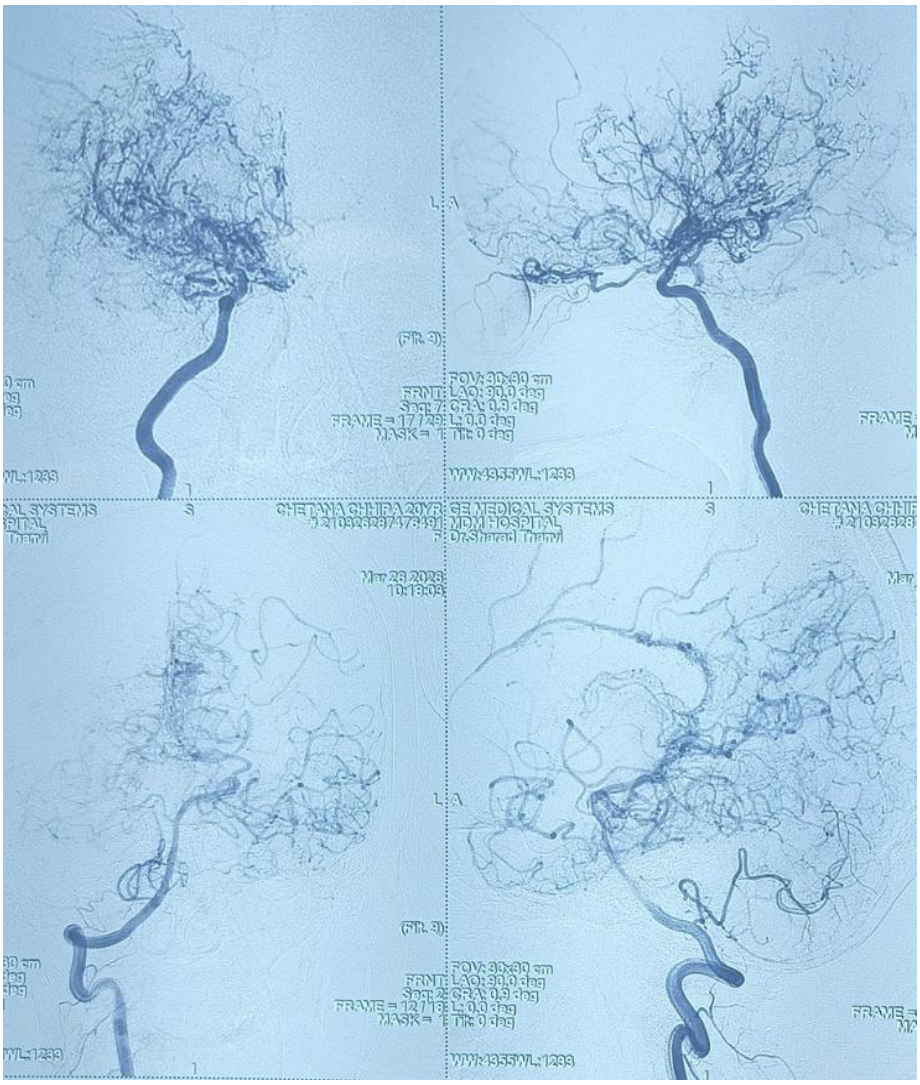


Figure 1: stenosis of distal ICA and multiple collaterals in posterior circulation and from external carotids



DSA images : classic puff of smoke appearance. Suggestive of Movamova disease

DISCUSSION

The present case series illustrates the remarkable clinical and radiological heterogeneity of MMD as encountered at a tertiary neurological centre in northern India. Our cohort of seven patients—predominantly young females (median age 20 years; female-to-male ratio 6:1)—aligns with the well-established epidemiological predilection of MMD for the younger female population.[4,11]

Ischaemic stroke was the most common acute presentation (Cases 5 and 6), consistent with the paediatric-predominant ischaemic phenotype described in the literature.[12] Notably, Case 6 (Rekha, 12 years) had experienced a prior episode 1 year before the index presentation, highlighting a critical missed-diagnosis window during which prompt revascularisation may have altered outcome. Intracranial haemorrhage, though characteristic of the adult phenotype in East Asian series, occurred in our sole male patient (Case 5, Narender, 34 years), in keeping with published Indian data wherein adult-onset MMD frequently manifests haemorrhagically.[13]

Three patients (Cases 1, 2, and 3) presented primarily with headache, one of the most common yet underappreciated manifestations of MMD. Headache in MMD likely reflects ischaemia-related cortical spreading depolarisation and meningeal stretch from dilated collaterals.[14] Recurrent migraine-type headache in a young woman, particularly when accompanied by even subtle focal or cognitive symptoms, should prompt cerebrovascular evaluation including MR angiography.

The coexistence of epilepsy as the primary presenting feature (Cases 4 and 2, the latter additionally) merits attention. Seizures in MMD are attributable to cortical ischaemia, gliosis, and in Case 4, hippocampal sclerosis secondary to chronic hemispheric underperfusion—a mechanism analogous to mesial temporal epileptogenesis in other ischaemic contexts.[15] Multi-drug anti-epileptic regimens were required alongside antiplatelet therapy.

Case 7 (Sahiba Bano) represents the only case of syndromic moyamoya in our series, secondary to NF1—a well-documented association arising from RAS-MAPK pathway-mediated vascular smooth muscle proliferation causing characteristic fibromuscular dysplasia of cranial and cervical vessels.[16] The classical NF1 cutaneous stigmata (café-au-lait spots, neurofibromas, Lisch nodules, axillary freckling, scoliosis) were conspicuously present, underscoring the importance of a thorough dermatological and systemic examination in all patients with moyamoya pattern angiography. Distinguishing NF1-associated moyamoya syndrome from idiopathic MMD is clinically meaningful, as the genetic counselling implications and surgical revascularisation response may differ.[17]

All patients underwent extensive secondary workup to exclude autoimmune arteritis (ANA, ENA, ANCA), haematological causes (sickle cell disease, thalassaemia—relevant in our Rajasthan-based cohort given the regional prevalence of haemoglobinopathies), and metabolic vasculopathy (homocysteine). Elevated homocysteine in Case 2 (Chetana Chhipa) is a noteworthy finding; hyperhomocysteinaemia promotes endothelial injury and vascular occlusion and may be a co-factor or independent risk modifier in MMD pathogenesis.[18] Supplementation was instituted accordingly.

Regarding management, none of our patients underwent surgical revascularisation. In Case 1, the family explicitly declined surgery despite Stage 4 disease; the remaining patients either had disease insufficiently progressive at the time to warrant surgical referral, or were managed pending further workup. All received antiplatelet therapy (aspirin in varying doses), lipid-lowering therapy (atorvastatin), and, where appropriate, antiepileptic drugs, nimodipine, or headache prophylaxis. While surgical revascularisation—particularly indirect procedures such as EDAS in children—forms the cornerstone of long-term disease modification, medical management may stabilise symptoms and reduce ischaemic burden in select cases.[10,19]

Functional outcomes in our series were acceptable; five of seven patients (71.4%) achieved mRS ≤2 at the most recent follow-up. The least favourable outcome was in Case 6 (Rekha, mRS 3), the patient with recurrent ischaemic stroke and bilateral severe disease. This underscores the imperative of early diagnosis and timely intervention before cumulative ischaemic injury accrues.

Limitations of this series include its retrospective design, small sample size inherent to the rarity of the condition, variable follow-up duration, absence of neuropsychological assessment and perfusion imaging (which would add valuable functional data), and the lack of genetic testing (RNF213 variants) due to resource constraints at our centre. Prospective, multicentre Indian registries for MMD are warranted to characterise the burden and phenotypic spectrum in the South Asian population more precisely.

CONCLUSION

Moyamoya disease in the Indian clinical setting manifests with significant heterogeneity—encompassing ischaemic stroke, intracranial haemorrhage, headache, and epilepsy—predominantly in young women. The recognition of syndromic associations such as NF1, thorough exclusion of secondary causes through autoimmune and haematological workup, and individualised medical or surgical management are the pillars of appropriate care. Clinicians across neurology, emergency medicine, and general practice should maintain a high index of suspicion for MMD in young patients presenting with unexplained cerebrovascular events, recalcitrant migraine, or refractory epilepsy, to minimise diagnostic delays and optimise long-term outcomes.

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