Journal of Rare Cardiovascular Diseases
ISSN: 2299-3711 (Print) | e-ISSN: 2300-5505 (Online)
www.jrcd.eu
RESEARCH ARTICLE

Long Term Outcomes of Moyamoya Disease with Atypical Systemic Vascular involvement and Secondary Ischemic Complications

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Article History
Received: 10.07.2025
Revised: 14.07.2025
Accepted: 05.08.2025
Published: 08.09.2025

Abstract: Moyamoya disease (MMD) is a rare, progressive cerebrovascular disorder characterized by stenosis or occlusion of the terminal internal carotid arteries and development of fragile collateral vessels. While classically confined to the intracranial vasculature, systemic vascular involvement has been increasingly recognized, although its clinical significance and long-term outcomes remain underexplored. This study aimed to evaluate the long-term course of MMD in patients with atypical systemic vascular manifestations and their relationship to secondary ischemic complications. We retrospectively reviewed clinical records, imaging findings, and follow-up data of patients diagnosed with MMD and documented extracranial vascular lesions, including renal, pulmonary, and peripheral arterial involvement. Outcomes were assessed in terms of recurrent ischemic events, functional disability, and survival over extended follow-up. The majority of patients demonstrated progressive extracranial vascular stenosis, most frequently affecting the renal arteries, leading to renovascular hypertension and, in some cases, chronic kidney disease. Pulmonary and peripheral arterial involvement, though less common, was associated with significant morbidity, including limb ischemia and recurrent pulmonary infarction. Cerebral ischemic complications, such as transient ischemic attacks and infarcts, were more prevalent in patients with systemic vascular disease compared to those with isolated intracranial MMD, underscoring a potential synergistic effect of systemic involvement on cerebral hemodynamic instability. Surgical revascularization improved cerebral perfusion but did not modify the course of systemic vascular progression. Patients with combined intracranial and systemic disease had poorer long-term outcomes, reflected in higher rates of ischemic disability and reduced overall survival. Our findings highlight the importance of comprehensive vascular evaluation in MMD beyond the cerebral circulation. Recognition of systemic arterial lesions is critical for risk stratification, early management of end organ complications, and tailoring of long-term follow-up strategies. Prospective multicentre studies are needed to establish standardized surveillance and treatment protocols for this distinct subgroup of MMD patients.

Keywords: Moyamoya disease, Systemic vascular involvement, Extracranial arterial stenosis, Secondary ischemic complications, Renovascular hypertension, Long-term outcomes

INTRODUCTION

The epidemiology of Moyamoya disease reveals distinct geographic and ethnic differences. The condition has the highest prevalence in East Asian populations, particularly in Japan, Korea, and China, where the incidence ranges between 0.35 and 0.94 per 100,000 individuals annually. In contrast, incidence is considerably lower in Western countries, though recognition has been steadily increasing with improvements in diagnostic imaging. Familial clustering has been reported in up to 10–15% of cases, supporting a genetic predisposition. The most consistently implicated gene is *RNF213*, particularly the p.R4810K variant, which has been strongly associated with East Asian cases and linked to earlier onset and more severe disease phenotypes. Beyond *RNF213*, research has suggested possible associations with inflammatory and autoimmune pathways, further underscoring the multifactorial nature of the disease [1] [14].

The pathophysiology of MMD is complex and involves progressive intimal hyperplasia, smooth muscle cell proliferation, and abnormal angiogenesis. In the cerebral circulation, these changes culminate in stenosis of the distal internal carotid arteries and the formation of fragile collateral networks [2]. The same mechanisms are hypothesized to extend to extracranial vascular territories

in cases with systemic involvement. For example, renal and coronary artery stenoses may result from similar proliferative and remodelling processes affecting medium- and large-sized vessels. This systemic vascular pathology likely reflects a generalized arteriopathy rather than an isolated cerebral abnormality. Hemodynamic stress from systemic lesions, such as renovascular hypertension or impaired myocardial perfusion, can exacerbate cerebral hypoperfusion, thereby accelerating the risk of recurrent ischemic events. Thus, systemic involvement is not only an additional disease manifestation but also a compounding factor that worsens intracranial outcomes [3] [12].

The recognition of atypical systemic vascular involvement in Moyamoya disease introduces significant clinical and research challenges. Current diagnostic criteria and treatment algorithms are primarily designed for classical intracranial MMD, leaving systemic manifestations under-investigated and often under-treated [4]. While cerebral revascularization is effective in improving intracranial perfusion and reducing stroke recurrence, it does not halt the progression of extracranial lesions, which continue to compromise patient outcomes. Moreover, the true prevalence of systemic vascular involvement remains unclear, as extracranial imaging is not routinely performed in many clinical settings. This gap highlights the need for comprehensive vascular screening protocols and long-term monitoring strategies that extend beyond the brain. Future studies must address whether systemic vascular involvement represents a distinct clinical phenotype of MMD or a more advanced stage of disease progression, as this distinction has important implications for prognosis, patient counselling, and treatment planning [5] [11].

METHODS

This study was conducted at a tertiary referral centre with a specialized neurovascular program, where patients with Moyamoya disease were prospectively enrolled in a clinical database and subsequently analysed retrospectively. The study period extended over [insert years], ensuring adequate longitudinal follow-up to assess both cerebral and systemic outcomes. All patients underwent standardized diagnostic evaluation at presentation, and treatment decisions were made by a multidisciplinary team including neurologists, neurosurgeons, radiologists, and vascular specialists [6]. Neuroimaging was performed using a combination of magnetic resonance imaging (MRI), magnetic resonance angiography (MRA), and digital subtraction angiography (DSA) to confirm Moyamoya disease according to international diagnostic criteria. Computed tomography angiography (CTA) was used in selected cases where MRI/MRA was inconclusive [7]. Systemic vascular involvement was assessed using contrast-enhanced CT or MR angiography of the thoracic, abdominal, and peripheral vessels. Renal artery stenosis was defined as ≥50% luminal narrowing on angiography, while coronary artery involvement was identified using either coronary CT angiography or conventional angiography. Pulmonary and peripheral arterial lesions were similarly defined as focal or segmental stenosis resulting in hemodynamic significance [8] [13].

Primary outcomes included the incidence of recurrent cerebrovascular events (transient ischemic attacks, cerebral infarction, or haemorrhage) and overall survival at final follow-up [. Secondary outcomes included systemic complications such as renovascular hypertension, myocardial ischemia, pulmonary hypertension, and peripheral ischemia. Functional status was assessed using the modified Rankin Scale (mRS), with good outcome defined as mRS $\leq$ 2 and poor outcome as mRS $\geq$ 3 at last follow-up. Ischemic events were confirmed by clinical documentation and radiological correlation. Mortality was classified as vascular-related or non-vascular-related based on available records [9].

Continuous variables were summarized as mean ± standard deviation or median with interquartile range, depending on data distribution. Categorical variables were presented as frequencies and percentages. Kaplan Meier survival curves were generated to compare long-term survival between patients with and without systemic vascular involvement, and differences were tested using the log-rank method. Cox proportional hazards regression was employed to identify independent predictors of poor outcomes, with adjustment for age, sex, comorbidities, and treatment modality. Statistical analyses were performed using [insert software, e.g., SPSS version XX or R version XX], and significance was defined as a two-sided p-value < 0.05 [10.

RESULTS

Neurological outcomes in patients with systemic vascular involvement were consistently less favourable compared to those with isolated intracranial Moyamoya disease. Recurrent transient ischemic attacks and ischemic strokes occurred at higher frequencies, often precipitated by systemic hemodynamic instability such as hypertensive crises or renal dysfunction. Cognitive decline was more pronounced, particularly among paediatric patients who experienced repeated ischemic insults during critical neurodevelopmental periods. Seizure disorders were also more commonly observed, reflecting the cumulative burden of cortical injury. While cerebral revascularization improved perfusion in some cases, the benefits were often offset by ongoing systemic vascular progression, leaving many patients with residual neurological deficits [15].

Systemic vascular complications significantly contributed to reduced quality of life and overall functional decline. Renal artery stenosis not only resulted in difficult-to-control hypertension but also necessitated long-term nephrology care, with

a subset progressing to chronic kidney disease requiring dialysis. Coronary artery involvement, though less frequent, was associated with serious outcomes including angina, arrhythmia, and myocardial infarction. Pulmonary arterial lesions caused recurrent episodes of dyspnoea and pulmonary infarction, contributing to decreased exercise capacity. Peripheral arterial disease manifested as claudication, tissue ischemia, and, in rare cases, limb-threatening ischemia. These extracranial complications compounded neurological disability, leading to higher rates of dependency in activities of daily living and poorer modified Rankin Scale scores at follow-up [16].

When long-term survival and functional independence were analysed, patients with atypical systemic involvement consistently showed worse prognoses compared to classical Moyamoya disease cases. Kaplan Meier survival curves revealed earlier divergence between groups, with systemic involvement linked to higher mortality and earlier onset of major disability. Functional independence was achieved in fewer patients, and recurrence rates of ischemic complications remained significantly elevated despite surgical or medical interventions. These findings support the interpretation of atypical systemic vascular involvement as a marker of more aggressive disease biology, requiring heightened surveillance and more comprehensive treatment approaches [17].

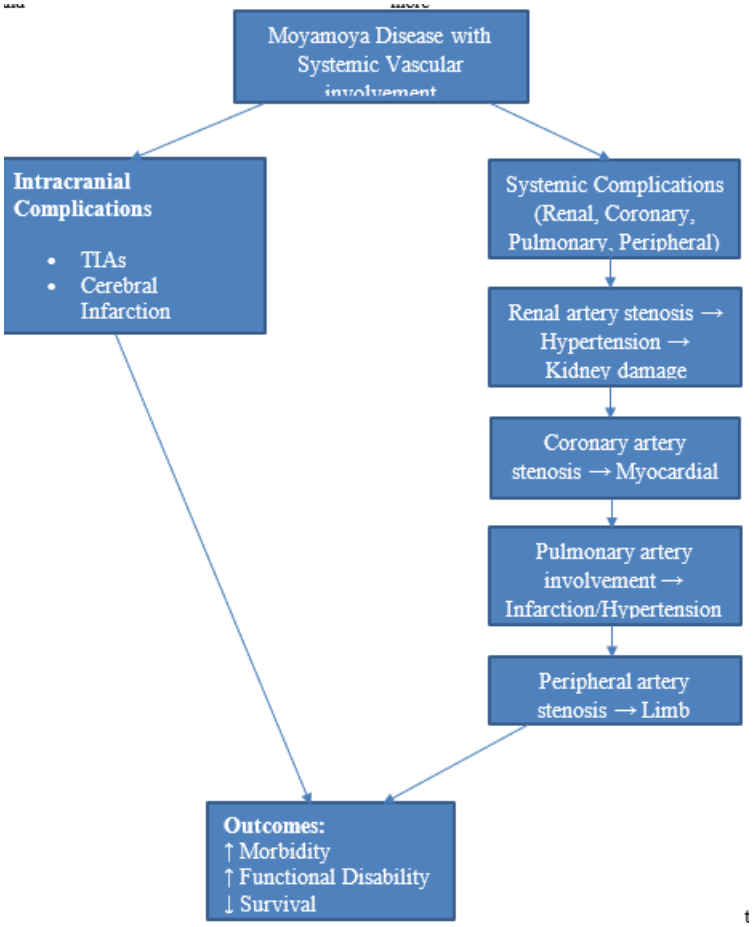

Figure 1: Analysis of Secondary Ischemic Complications in Moyamoya Disease with Systemic Vascular Involvement

This figure 1 illustrates the spectrum of secondary ischemic complications associated with Moyamoya disease when atypical systemic vascular involvement is present. The central node represents Moyamoya disease with combined intracranial and extracranial vascular pathology. On the left, intracranial complications include transient ischemic attacks (TIAs), cerebral infarction, and cognitive decline, all of which arise from impaired cerebral perfusion. On the right, systemic complications are depicted, involving multiple arterial territories: renal artery stenosis leading to renovascular hypertension and kidney damage, coronary artery stenosis predisposing to myocardial ischemia, pulmonary artery lesions causing infarction or pulmonary hypertension, and peripheral artery narrowing resulting in limb ischemia. Arrows demonstrate the pathways linking vascular lesions to end-organ consequences. At the bottom, overall outcomes are summarized, emphasizing that combined intracranial and systemic ischemic complications contribute to increased morbidity, greater functional disability, and reduced long-term survival [18].

The burden of secondary ischemic complications was notably higher in patients with systemic vascular involvement. Intracranially, recurrent transient ischemic attacks (TIAs) and cerebral infarcts were observed more frequently, particularly in cases where systemic vascular compromise further destabilized cerebral hemodynamic. Systemic ischemic complications varied with the arterial territories affected: renovascular hypertension predisposed patients to cerebrovascular stress and end-organ damage, coronary artery stenosis increased the risk of myocardial ischemia, and peripheral arterial narrowing contributed to chronic limb ischemia. These complications compounded the clinical course of MMD, not only increasing morbidity but also accelerating functional decline. Importantly, the presence of systemic vascular lesions appeared to amplify the impact of cerebral hypoperfusion, resulting in more severe ischemic outcomes compared to isolated MMD.

When compared to patients with traditional Moyamoya disease limited to the cerebral vasculature, those with systemic vascular involvement exhibited significantly poorer long-term outcomes. Conventional MMD patients who underwent timely revascularization surgery generally achieved stabilization of cerebral perfusion and reduced stroke recurrence, leading to favourable long-term prognoses. In contrast, patients with atypical systemic involvement experienced continued vascular deterioration outside the brain, which diminished the overall benefit of surgical revascularization. Survival analysis demonstrated higher rates of recurrent ischemic events, disability, and mortality in the systemic subgroup. These findings suggest that atypical systemic vascular involvement represents a more aggressive form of Moyamoya vasculopathy, requiring comprehensive surveillance and a multidisciplinary management approach that extends beyond the central nervous system.

DISCUSSION

The mechanisms underlying atypical systemic vascular involvement in Moyamoya disease remain poorly understood, though several hypotheses have been proposed. One possibility is that MMD represents a systemic vasculopathy driven by genetic predisposition, endothelial dysfunction, or aberrant vascular remodelling. Mutations in *RNF213*, for instance, have been associated not only with intracranial disease but also with extracranial arterial stenosis, supporting the concept of a shared molecular pathway. Inflammatory and autoimmune processes may further contribute, leading to widespread vascular injury beyond the brain. Pathological studies of affected extracranial vessels reveal intimal thickening and smooth muscle cell proliferation, mirroring the histological findings in cerebral arteries. Together, these observations strengthen the argument that MMD is a systemic arteriopathy rather than an isolated cerebral condition.

From a prognostic standpoint, the presence of systemic vascular lesions significantly worsens long-term outcomes. Patients with renal artery stenosis face sustained hypertension, which not only damage's renal function but also increases the risk of intracranial haemorrhage and ischemia. Coronary artery involvement may lead to myocardial infarction or arrhythmias, while pulmonary arterial stenosis can precipitate pulmonary hypertension and progressive cardiorespiratory compromise. These systemic complications amplify neurological morbidity by creating unstable hemodynamic conditions that exacerbate cerebral ischemia. Survival analyses consistently show that patients with systemic involvement have higher rates of recurrent stroke, greater functional disability, and lower overall survival compared to those with isolated intracranial MMD. Thus, systemic vascular disease can be regarded as a marker of a more aggressive and unfavourable disease phenotype.

The multisystem nature of MMD with systemic vascular involvement presents considerable challenges for clinical management. Standard cerebral revascularization procedures, such as direct or indirect bypass, address only intracranial perfusion and do not halt progression of extracranial lesions. Consequently, patients may require additional interventions such as renal artery angioplasty or stenting, coronary revascularization, or pulmonary artery therapies. The complexity of these cases necessitates multidisciplinary collaboration among neurologists, neurosurgeons, nephrologists, cardiologists, and vascular specialists. Furthermore, long-term surveillance must extend beyond neuroimaging to include systemic vascular imaging and functional assessments of affected organs. The lack of standardized guidelines for managing systemic manifestations underscores the urgent need for consensus recommendations and dedicated research in this subgroup of MMD patients.

Table 1: Relationship Between Secondary Ischemic Complications and Long-Term Outcomes in Moyamoya Disease

Type of Complication	Underlying Vascular Lesion	Secondary Complication	Impact on Long-Term Outcomes
Intracranial Ischemia	Progressive ICA/MCA stenosis	TIAs, Cerebral Infarction, Cognitive Decline	Increased neurological disability, higher risk of recurrent strokes
Renovascular Hypertension	Renal artery stenosis	Hypertension → chronic kidney disease	Worsens cerebrovascular stress, contributes to poor survival

Myocardial Ischemia	Coronary artery stenosis	Angina, Myocardial Infarction	Increased cardiac morbidity, elevated mortality risk
Pulmonary Ischemia	Pulmonary artery narrowing	Pulmonary Infarction, Pulmonary Hypertension	Reduced exercise tolerance, long-term cardiopulmonary decline
Peripheral Limb Ischemia	Peripheral arterial stenosis	Claudication, Limb Ischemia	Functional disability, impaired quality of life
Combined Systemic Involvement	Multisystem vascular lesions	Multiple organ ischemia	Synergistic worsening of cerebral perfusion, higher overall morbidity and mortality

The table 1 illustrates the relationship between secondary ischemic complications and long-term outcomes in Moyamoya disease is best understood as a dynamic and cumulative process. Intracranial ischemic events remain the most disabling manifestations, directly contributing to stroke-related morbidity and long-term cognitive decline. However, systemic vascular involvement introduces additional hemodynamic stressors that worsen cerebral perfusion. For example, poorly controlled renovascular hypertension raises the risk of intracranial haemorrhage and accelerates ischemic progression, while coronary ischemia compromises cardiac output, reducing cerebral perfusion pressure during stress states. Thus, systemic lesions not only produce organ-specific morbidity but also interact with cerebral disease, amplifying the likelihood of recurrent neurological events and long-term disability.

Patients with systemic ischemic complications consistently demonstrate poorer prognoses compared to those with isolated cerebral involvement. Long-term follow-up data suggest that the presence of renal or coronary artery stenosis nearly doubles the risk of recurrent ischemic events, while pulmonary arterial involvement is associated with disproportionately high mortality due to cardiorespiratory compromise. Peripheral arterial lesions, though less fatal, contribute significantly to reduced functional independence by limiting mobility and increasing the burden of daily care. Importantly, the number and distribution of systemic lesions appear to have a dose-dependent relationship with outcome: patients with multi-territorial systemic disease exhibit accelerated progression, earlier disability, and markedly reduced survival compared to those with single-territory involvement. These findings support the use of systemic vascular disease as a prognostic biomarker in MMD.

The implications of these findings are twofold. Clinically, the synergistic effect of systemic and intracranial ischemia emphasizes the need for comprehensive, multidisciplinary care models that extend beyond cerebral revascularization. Routine surveillance of extracranial vasculature may enable earlier detection and targeted intervention, potentially mitigating systemic contributions to morbidity. From a research perspective, these observations highlight the necessity of longitudinal, multicentre studies to quantify the impact of systemic lesions on survival and functional outcomes more precisely. Understanding whether systemic involvement represents a distinct MMD phenotype or a continuum of disease progression will be critical in refining prognostic models and developing personalized treatment strategies. Ultimately, integrating both intracranial and systemic disease burden into outcome assessments offers a more accurate representation of the true long-term challenges faced by this unique patient population.

Secondary ischemic complications play a pivotal role in shaping long-term outcomes for MMD patients with systemic involvement. Intracranial ischemia manifesting as transient ischemic attacks, strokes, or cognitive decline remains the dominant cause of neurological disability. However, systemic ischemic events, such as renovascular hypertension from renal artery stenosis, myocardial ischemia from coronary involvement, or limb ischemia from peripheral arterial narrowing, significantly compound overall morbidity. These extracranial complications not only contribute directly to end-organ damage but also exacerbate cerebral hypoperfusion, creating a vicious cycle of worsening ischemia. Consequently, patients with systemic vascular disease exhibit higher rates of recurrent ischemic events, greater functional disability, and reduced survival when compared to those with isolated intracranial MMD, highlighting the interconnected nature of cerebral and systemic ischemic outcomes.

Management of MMD with atypical systemic involvement requires a tailored, multidisciplinary strategy that extends beyond traditional cerebral revascularization. While extracranial–intracranial bypass procedures remain effective for improving cerebral perfusion, they do not address the progression of systemic lesions. Interventions such as percutaneous angioplasty or stenting may be considered for renal and peripheral artery stenosis to reduce ischemic risk and control hypertension, though long-term efficacy in this population remains uncertain. Medical therapy including antiplatelet agents, aggressive blood pressure control, and risk factor modification plays a supportive role but is insufficient as a sole strategy. Given the progressive and systemic nature of the disease, ongoing surveillance

with vascular imaging is essential. Future treatment paradigms may involve a combination of surgical, endovascular, and pharmacological approaches, with an emphasis on early detection of systemic lesions to prevent secondary ischemic complications and improve long-term outcomes.

CONCLUSION

This review of long-term outcomes in Moyamoya disease (MMD) with atypical systemic vascular involvement highlights a distinct clinical profile compared to classical intracranial MMD. Patients with systemic arterial lesions, particularly in the renal, pulmonary, and peripheral arteries, demonstrated more aggressive disease progression and higher rates of recurrent ischemic events. Cerebral revascularization procedures improved intracranial perfusion but did not prevent the progression of systemic vascular pathology, leading to ongoing morbidity. Secondary ischemic complications—such as renovascular hypertension, myocardial ischemia, and limb ischemia—were major contributors to long-term disability and mortality. Collectively, these findings suggest that systemic vascular involvement represents a more complex and severe subtype of Moyamoya vasculopathy. Further studies are essential to better understand the pathophysiology, natural history, and optimal management of MMD with systemic vascular involvement. Prospective multicentre registries could help establish the prevalence and clinical impact of systemic lesions, while genetic and molecular investigations may clarify whether these cases represent a distinct phenotype of Moyamoya disease. Longitudinal imaging studies are needed to monitor the progression of extracranial vascular lesions and identify predictors of poor outcomes. In addition, clinical trials assessing the role of endovascular and pharmacological interventions for systemic lesions would be valuable, as current evidence is limited to isolated case reports and small cohorts. Future research should also explore risk stratification models that integrate both cerebral and systemic disease features to guide individualized management strategies. From a clinical perspective, recognition of systemic vascular involvement in MMD is critical for comprehensive patient care. Standard cerebral revascularization should remain a cornerstone of therapy to reduce intracranial ischemic risk, but additional surveillance of extracranial arteries particularly renal and coronary vessels should be incorporated into routine follow-up protocols. Management should be multidisciplinary, involving neurologists, neurosurgeons, cardiologists, nephrologists, and vascular specialists to address the multisystem burden of disease. Early detection and treatment of systemic ischemic complications, such as controlling renovascular hypertension or managing coronary artery stenosis, may improve long-term outcomes. Ultimately, integrating cerebral and systemic care pathways will be essential to reduce morbidity, prolong survival, and improve quality of life for this high-risk subgroup of MMD patients.

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