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

Comprehensive Assessment of Segmental Arterial Medialises as A Rare Cause of Systemic Circulatory Failure and Aneurysm Formation

Deepak Kumar Sahu[1] and Dr. Sushma Dubey[2]
[1]Assistant Professor, Department of Pharmacy, Kalinga University, Raipur, India.
[2]Assistant Professor, Department of Biotechnology, Kalinga University, Raipur, India.

*Corresponding Author
Deepak Kumar Sahu
(ku.deepakkumarsahu@kalingauniversity.ac.in)

Article History
Received: 14.08.2025
Revised: 25.08.2025
Accepted: 17.09.2025
Published: 30.09.2025

Abstract: Segmental arterial medialises (SAM) is an uncommon, non-atherosclerotic, non-inflammatory arteriopathy characterized by lytic degeneration of the arterial media, leading to acute vascular instability. Although rare, SAM carries significant clinical importance due to its potential to cause catastrophic systemic circulatory failure and aneurysm formation. The disease predominantly affects medium- to large-sized abdominal and visceral arteries, but extra-abdominal involvement has also been documented. The pathophysiology is hypothesized to involve vasospastic injury and subsequent apoptotic loss of smooth muscle cells within the media, resulting in arterial wall weakening. Clinically, SAM presents with a spectrum ranging from asymptomatic incidental vascular lesions to life-threatening haemorrhage, hypovolemic shock, or ischemic end-organ damage. Diagnostic evaluation is challenging, as SAM often mimics other vasculopathies such as fibromuscular dysplasia, systemic vasculitis, or connective tissue disorders. Imaging modalities including CT angiography and MR angiography typically reveal multifocal arterial dissections, aneurysms, or stenotic lesions with a segmental distribution, while histopathology remains the gold standard for definitive diagnosis. Importantly, laboratory investigations usually lack evidence of systemic inflammation, helping to differentiate SAM from vasculitis processes. Management strategies depend on clinical presentation and disease severity. Hemodynamically unstable patients may require urgent endovascular or surgical intervention to control haemorrhage or repair aneurysms, whereas stable patients can be managed conservatively with vigilant surveillance. Pharmacologic immunosuppression is ineffective given the non-inflammatory nature of SAM, and supportive care with blood pressure optimization remains a cornerstone of therapy. Recognition of SAM as a distinct pathological entity is crucial to avoid misdiagnosis and inappropriate treatment. A comprehensive assessment highlights its role as a rare but formidable cause of systemic circulatory failure and aneurysm formation. Improved awareness, early detection, and individualized therapeutic approaches are essential to reduce morbidity and mortality associated with this enigmatic vascular disorder.

Keywords: Segmental arterial medialises, non-atherosclerotic arteriopathy, systemic circulatory failure, aneurysm formation; visceral artery dissection, vascular pathology.

INTRODUCTION

The hallmark of segmental arterial medialises (SAM) lies in the vacuole degeneration and apoptosis of arterial smooth muscle cells within the media. This leads to focal gaps in the vessel wall, exposing the adventitia to high intraluminal pressures. Such changes predispose the vessel to dissecting hematomas, aneurysm formation, and ultimately rupture. Importantly, there is no evidence of inflammation or immune-mediated injury, distinguishing SAM from vasculitis's. Although the exact ethology remains uncertain, vasospastic injury mediated by catecholamine surges, stress-related insults, or exogenous vasoactive agents has been proposed [1]. These stimuli may trigger ischemic injury to the vasa vasorum and apoptosis of medial smooth muscle cells. Genetic predispositions or underlying connective tissue fragility may also play permissive roles, though strong evidence is lacking. The disease is considered biphasic: an acute injurious phase characterized by lytic medial degeneration, followed by a reparative phase marked by fibrous scarring and vessel remodelling [2].

The pathophysiological cascade in SAM directly contributes to systemic circulatory failure when vessel rupture leads to catastrophic haemorrhage and hypovolemic shock. Similarly, flow-limiting dissections or occlusions can precipitate ischemic organ dysfunction, further aggravating circulatory compromise [3]. Understanding these mechanisms is critical, as early

recognition of arterial instability may prompt life-saving interventions.

Patients with SAM may present with nonspecific or acute symptoms depending on the arteries involved. Common manifestations include sudden abdominal pain, flank pain, or chest pain due to intra-abdominal or intrathoracic arterial involvement. In severe cases, rupture of visceral artery aneurysms can lead to hypovolemic shock, requiring emergent resuscitation. Visceral involvement is most common, particularly in the celiac, superior mesenteric, renal, and splenic arteries. Rupture may cause intra-abdominal haemorrhage, while dissections can lead to mesenteric ischemia, renal infarction, or hepatic dysfunction. Extra-abdominal involvement, though less frequent, can manifest with stroke, myocardial ischemia, or peripheral ischemia depending on the affected vascular territory [4]. The nonspecific presentation and rarity of SAM often result in delayed recognition or misdiagnosis. Many patients are initially treated for vasculitis or other vascular disorders. The overlap in clinical and radiological features further complicates accurate diagnosis. This underscores the importance of maintaining a high index of suspicion in patients presenting with spontaneous intra-abdominal haemorrhage or unexplained circulatory collapse. Computed tomography angiography (CTA) is the most useful diagnostic tool, revealing characteristic findings such as arterial dissections, fusiform aneurysms, stenoses, or arterial wall irregularities distributed in a segmental fashion. Magnetic resonance angiography (MRA) and digital subtraction angiography (DSA) may provide complementary detail, particularly in pre-intervention planning [5].

Definitive diagnosis requires histological confirmation, though tissue samples are rarely available due to the emergent nature of presentation. Histopathology demonstrates medialises, absence of inflammation, adventitial expansion, and dissecting hematomas. The lack of inflammatory infiltrates is the most important differentiating factor from vasculitis [6]. SAM is frequently confused with fibromuscular dysplasia, systemic vasculitis, connective tissue disorders (e.g., Ehlers–Danlos syndrome), or infectious arteritis. Unlike these conditions, SAM lacks systemic inflammatory markers, autoantibody positivity, or systemic connective tissue features. Correct differentiation is essential to avoid inappropriate treatment with immunosuppressants, which offer no benefit in SAM.

BACKGROUND

Segmental arterial medialises (SAM) was first described in 1976 by Slaving and Gonzalez-Vitale, who characterized it as a distinct, non-inflammatory vasculopathy involving medium- to large-sized arteries. Initially, the condition was often misclassified as arteritis due to its destructive vascular changes, but the absence of inflammatory infiltrates on histology established it as a separate entity [7]. Over subsequent decades, case reports and small series have broadened the understanding of SAM, highlighting its predilection for abdominal visceral arteries such as the celiac, superior mesenteric, and renal arteries. Despite advancements in imaging and vascular pathology, SAM remains underdiagnosed due to its rarity, variable presentation, and tendency to mimic other vascular disorders like fibromuscular dysplasia or systemic vasculitis [8].

The hallmark of SAM is non-inflammatory lytic degeneration of the arterial media. This process begins with apoptosis and vacuolization of smooth muscle cells, leading to focal medial defects and disruption of the structural integrity of the vessel wall [9]. These defects allow intramural haemorrhage, dissection, and aneurysm formation. The ethology is not fully understood, but hypotheses suggest that vasoactive stimuli, including catecholamine surges or exogenous vasoconstrictors, trigger ischemic injury in the vasa vasorum, initiating medial cell death. The disease typically progresses through two stages: an acute injurious phase marked by medialises and dissection, followed by a reparative phase characterized by fibrosis and vessel remodelling. This unique pathophysiology distinguishes SAM from inflammatory vasculitis's and genetic arteriopathies [10].

SAM poses a significant risk for systemic circulatory failure due to its capacity to induce catastrophic vascular events. Rupture of visceral or thoracic artery aneurysms may cause massive haemorrhage and hypovolemic shock, rapidly overwhelming circulatory homeostasis. Similarly, dissections or occlusions resulting from medial disruption can compromise blood flow to vital organs, precipitating ischemia and multiorgan dysfunction. In some patients, the abrupt onset of haemorrhage or ischemia may present as sudden cardiovascular collapse with few preceding warning signs. Recognizing SAM as a potential ethology of circulatory failure is crucial, as timely diagnosis and targeted management can mean the difference between survival and mortality [11].

Clinical Presentation

The clinical manifestations of segmental arterial medialises (SAM) are diverse, ranging from asymptomatic incidental findings to life-threatening vascular emergencies. The most common presenting symptom is acute, severe abdominal or flank pain, which typically results from dissection or rupture of visceral arteries such as the celiac, superior mesenteric, or renal arteries [12]. In catastrophic cases, arterial rupture leads to massive intra-abdominal or retroperitoneal haemorrhage, presenting with hypotension, tachycardia, and hypovolemic shock. Other symptoms depend on the vascular territory affected: renal involvement may cause flank pain or haematuria, mesenteric ischemia can result in abdominal angina or bowel infarction, and thoracic artery lesions may cause chest pain or hemithorax. Extra-abdominal involvement, though

rare, can lead to neurological deficits from carotid or cerebral artery dissections, or myocardial ischemia if coronary arteries are involved [14].

Currently, no universally accepted diagnostic criteria exist for SAM, and diagnosis often requires a combination of clinical suspicion, imaging findings, and histopathological confirmation when feasible [13]. Radiological hallmarks on computed tomography angiography (CTA) or magnetic resonance angiography (MRA) include multifocal arterial dissections, fusiform aneurysms, stenoses, or arterial wall irregularities in a segmental distribution. Digital subtraction angiography (DSA) may further delineate arterial lesions and guide therapeutic interventions. Laboratory investigations are typically unremarkable, with normal inflammatory markers and negative autoantibody profiles, which help differentiate SAM from systemic vasculitis. Histopathology, though rarely available due to the emergent nature of presentation, remains the gold standard, showing medialises of the arterial media without inflammatory infiltrates.

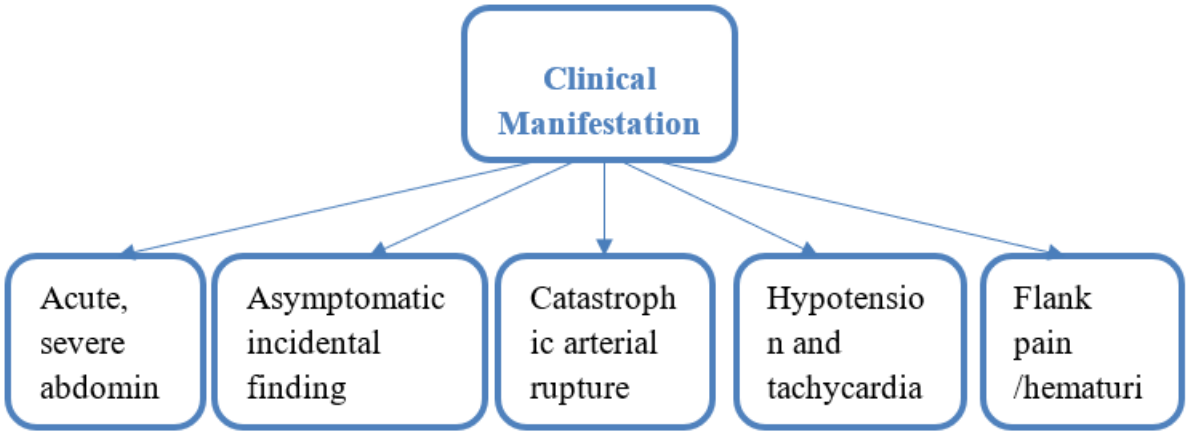

Figure 1: Clinical Manifestations of Segmental Arterial Medialises (SAM)

This figure 1 illustrates the spectrum of clinical manifestations in Segmental Arterial Medialises (SAM). The central category of "Clinical Manifestations" branches into common presentations such as asymptomatic incidental findings, acute severe abdominal pain, catastrophic arterial rupture, hemodynamic compromise with hypotension and tachycardia, and renal involvement manifested as flank pain or haematuria [15]. These signs often reflect arterial dissection, aneurysm, or rupture within visceral vessels such as the celiac, mesenteric, or renal arteries, making them critical red flags for early recognition of SAM.

In addition to these primary features, SAM may extend to other vascular territories, causing diverse systemic effects [16]. Mesenteric ischemia can result in abdominal angina or bowel infarction, thoracic artery lesions may present with chest pain or hemithorax, carotid or cerebral dissections can produce neurological deficits, and coronary artery involvement may trigger myocardial ischemia. Together, these manifestations underscore the heterogeneity and severity of SAM, ranging from silent incidental findings to catastrophic vascular emergencies, reinforcing the importance of high clinical suspicion and prompt diagnostic evaluation.

Treatment and Management

Management of segmental arterial medialises (SAM) depends on the severity of presentation and the stability of the patient. In hemodynamically stable patients without active bleeding, conservative therapy is often pursued, including strict blood pressure control to reduce the risk of aneurysm rupture and close radiological surveillance to monitor disease progression. Anticoagulation and immunosuppressive therapy are generally avoided, as SAM is non-inflammatory in nature and lacks a thrombotic basis. In cases of active haemorrhage, endovascular approaches such as coil embolization, stent grafting, or vessel occlusion are considered first-line interventions, given their minimally invasive nature and ability to rapidly control bleeding [17].

Table 1: Clinical Features, Diagnostic Criteria, and Differential Diagnosis of Segmental Arterial Medialises (SAM)

Category	Subcategory	Description / Findings	Clinical Relevance / Notes
Symptoms	Abdominal Pain	Sudden, severe, can be localized to flank or epigastrium	Most common presenting symptom; may indicate arterial rupture
	Nausea & Vomiting	Associated with abdominal pain	Non-specific but common

	Hypotension / Shock	If intra-abdominal haemorrhage occurs	Indicates urgent need for intervention
	Ischemic Symptoms	Rare; organ-specific depending on artery involved	Suggests arterial occlusion or dissection
	Incidental Findings	Sometimes asymptomatic; found on imaging	Often in patients imaged for unrelated reasons
Diagnostic Criteria	Imaging	CT angiography, MR angiography, conventional angiography	Reveals aneurysms, dissections, or segmental arterial dilatation
	Histopathology	Medialise of arterial media; no inflammation or atherosclerosis	Confirms diagnosis if tissue is available
	Laboratory Findings	Generally nonspecific; inflammatory markers usually normal	Helps differentiate from vasculitis
	Exclusion of Other Causes	Rule out atherosclerosis, vasculitis, infections	Essential to establish SAM diagnosis
Differential Diagnosis	Inflammatory Vasculitis	Polyarteritis nodosa, etc.; systemic inflammation, lab markers positive	SAM lacks systemic inflammation
	Atherosclerosis	Chronic plaque formation, calcification	SAM typically acute and non-atherosclerotic
	Fibromuscular Dysplasia (FMD)	"String-of-beads" appearance, usually younger patients	Rarely causes acute haemorrhage like SAM
	Mycotic / Infectious Aneurysm	Associated with infection, fever, positive cultures	SAM is non-infectious
	Connective Tissue Disorders	Ehlers-Danlos, Marfan; systemic signs, genetic confirmation	May mimic aneurysms but different systemic involvement
Other Features	Recurrence	Rare after acute phase	Helps differentiate from chronic vasculitis
	Prognosis	Variable; dependent on complications	Early detection critical for survival

The table **1** provides a structured overview of how SAM presents, is diagnosed, and differentiated from other vascular conditions. The first section focuses on symptoms**,** highlighting that SAM often manifests with sudden abdominal or flank pain, nausea, vomiting, or hypotension in cases of intra-abdominal haemorrhage. Some patients may present with ischemic symptoms depending on arterial involvement, while others are asymptomatic and discovered incidentally. This section emphasizes the variable and sometimes acute nature of SAM, which can pose diagnostic challenges [18].

The second part of the table outlines diagnostic criteria and differential diagnosis. Diagnosis primarily relies on imaging findings such as arterial aneurysms, dissections, or segmental dilatations, supported by histopathology showing medialises of the arterial media without inflammation or atherosclerosis. Laboratory markers are generally nonspecific, helping to distinguish SAM from inflammatory vasculitis.

The table also lists key differential diagnoses, including vasculitis, atherosclerosis, fibromuscular dysplasia, infectious aneurysms, and connective tissue disorders, each with distinguishing clinical or imaging features. Finally, additional features such as recurrence risk and prognosis are included, underlining that SAM is usually non-progressive, and long-term outcomes are favourable when complications are managed promptly.

The prognosis of SAM is highly variable and depends largely on the presence of complications such as rupture, ischemia, or multiorgan dysfunction. Mortality rates remain significant in cases of massive intra-abdominal haemorrhage, particularly if diagnosis and intervention are delayed. However, patients who survive the acute phase often experience stabilization or even regression of arterial lesions during the reparative phase. Long-term outcomes are generally favourable with proper blood pressure management and periodic imaging surveillance to detect recurrent or new vascular lesions. Importantly, unlike inflammatory vasculitis's, SAM does not appear to be a chronic progressive disease, and recurrences are rare once the acute phase has resolved.

Future therapeutic strategies for SAM are likely to focus on earlier recognition, individualized vascular protection, and advanced endovascular technologies. Improvements in high-resolution imaging may allow earlier detection of subtle arterial wall changes before catastrophic rupture occurs. Novel endovascular devices, such as bioresorbable scaffolds or flow-diverting stents, may provide safer, longer-lasting vascular stabilization compared to current methods. In addition, further research into the molecular mechanisms underlying medial smooth muscle apoptosis could open the door for targeted pharmacological interventions that prevent or reverse the destructive phase of the disease. Multicentre registries

and collaborative studies are essential to better define optimal management pathways, as the current evidence base is largely limited to case reports and small series.

CASE STUDIES

Case reports and small series have provided much of the clinical understanding of segmental arterial medialises (SAM), given its rarity. One illustrative case involved a 55-year-old man presenting with sudden abdominal pain and hypovolemic shock due to rupture of a celiac artery aneurysm. Another patient, a 42-year-old woman, was found to have bilateral renal artery dissections after complaining of flank pain and hypertension, without laboratory evidence of vasculitis. More atypical cases include elderly patients with spontaneous intracranial haemorrhage linked to cerebral artery involvement, and younger patients with coronary or mesenteric artery dissections. These diverse presentations highlight the unpredictable nature of SAM, which can manifest either catastrophically with rupture or more indolently with ischemia-related symptoms.

Treatment strategies in these cases depended largely on the acuity of presentation and vascular territory involved. The patient with a ruptured celiac artery aneurysm underwent urgent endovascular coil embolization, which successfully controlled bleeding and stabilized circulation. The woman with renal artery dissections was managed conservatively with antihypertensive therapy and close imaging follow-up, showing gradual stabilization of arterial lesions over several months. In cases involving cerebral or coronary arteries, stent placement or bypass surgery was employed due to the critical nature of perfusion in these regions. Outcomes were variable: while most patients survived with appropriate and timely intervention, mortality remained high in those presenting with massive intra-abdominal haemorrhage and delayed recognition.

Several important lessons emerge from published SAM cases. First, clinical suspicion is essential when patients present with unexplained intra-abdominal bleeding or spontaneous arterial dissections without systemic inflammatory markers. Second, radiological evaluation particularly computed tomography angiography (CTA) is invaluable for rapid diagnosis, as it demonstrates hallmark findings such as multifocal dissections, aneurysms, and stenoses in a segmental distribution. Third, the importance of tailoring management cannot be overstated: stable patients often benefit from conservative measures, while those with life-threatening haemorrhage require urgent endovascular or surgical therapy. Finally, inappropriate use of immunosuppressants should be avoided, as SAM is non-inflammatory, and such therapy delays definitive intervention.

The collective evidence from case studies underscores the need for heightened awareness of SAM as a cause of systemic circulatory failure and aneurysm formation. These cases reveal that while SAM is rare, its outcomes are highly dependent on early recognition and rapid, context-specific treatment. They also highlight the importance of multidisciplinary care, involving radiologists, vascular surgeons, and critical care specialists. Moreover, recurring themes such as misdiagnosis as vasculitis, the reliance on imaging for detection, and the success of endovascular interventions point toward clear strategies for improving patient survival. Together, these lessons provide a foundation for developing more standardized diagnostic criteria and treatment guidelines in the future.

CONCLUSION

This paper has explored segmental arterial medialises (SAM) as a rare but clinically significant vascular disorder. SAM is characterized by non-inflammatory lytic degeneration of the arterial media, which predisposes affected vessels to aneurysm formation, dissection, and rupture. Clinical manifestations vary widely, ranging from acute intra-abdominal haemorrhage to subtle ischemic symptoms depending on the arteries involved. Case studies illustrate the heterogeneity of presentation and underscore the critical role of imaging modalities, particularly computed tomography angiography, in diagnosis. Management strategies are tailored according to the patient's hemodynamic stability, including conservative monitoring, endovascular interventions, or surgical repair. Despite increasing recognition, SAM remains under-researched, with most knowledge derived from case reports and small case series. Future research should focus on multicentre registries to gather comprehensive clinical, radiological, and histopathological data. Molecular studies exploring the mechanisms of smooth muscle apoptosis and medial degeneration may identify therapeutic targets to prevent vascular compromise. Additionally, prospective studies are needed to clarify the natural history, recurrence rates, and long-term outcomes of SAM, as well as to develop standardized diagnostic criteria and risk stratification tools for clinical practice. Awareness of SAM as a cause of systemic circulatory failure has significant clinical implications. Early recognition allows timely interventions, preventing catastrophic haemorrhage and organ ischemia. Understanding the pathophysiology and clinical course of SAM guides appropriate treatment decisions, such as distinguishing patients who can be managed conservatively from those requiring urgent endovascular or surgical intervention. Moreover, recognizing SAM prevents misdiagnosis and unnecessary immunosuppressive therapy, which would be ineffective and potentially harmful. In summary, SAM represents a rare but critical ethology of vascular instability and systemic circulatory failure. Key lessons from the literature and case studies emphasize the importance of clinical vigilance, rapid imaging-based diagnosis, and individualized treatment approaches.

Improved understanding of SAM not only aids in the management of acute vascular emergencies but also contributes to broader knowledge of non-inflammatory arteriopathies. Future research and collaboration will be essential to establish evidence-based guidelines, optimize patient outcomes, and reduce morbidity and mortality associated with this enigmatic vascular disorder.

REFERENCES

1. Mehra, P., & Patel, K. (2024). A Metrics Driven Approach to Brand Management and Brand Health Check. In *Brand Management Metrics* (pp. 31-47). Periodic Series in Multidisciplinary Studies.
2. Malhotra, A., & Joshi, S. (2025). Exploring the Intersection of Demographic Change and Healthcare Utilization: An Examination of Age-Specific Healthcare Needs and Service Provision. *Progression Journal of Human Demography and Anthropology*, *3*(1), 8-14.
3. Basanta Kumar, R., & Sunil, K. (2024). Biotechnological Approaches to Develop Personalized Medicines for Rare Genetic Disorders. *Clinical Journal for Medicine, Health and Pharmacy*, *2*(2), 20-28.
4. Nair, M., & Rao, A. (2023). Blockchain for Terminology Traceability in Decentralized Health Systems. *Global Journal of Medical Terminology Research and Informatics*, *1*(1), 9-11.
5. Reddy, S. S. R. D., & Ramanadham, U. K. (2017). Big data analytics for healthcare organization, BDA process, benefits and challenges of BDA: a review. Advances in Science Technology and Engineering Systems Journal, 2(4), 189-196.
6. Durga, R., & Sudhakar, P. (2015). Design of a Wireless Transfer for a Secure Signal of Sender and Receiver System through the Network. International Journal of Applied Engineering Research, 10(82).
7. Fu, W., & Zhang, Y. (2025). The role of embedded systems in the development of smart cities: A review. SCCTS Journal of Embedded Systems Design and Applications, 2(2), 65–71.
8. Surendar, A. (2024). Internet of medical things (IoMT): Challenges and innovations in embedded system design. SCCTS Journal of Embedded Systems Design and Applications, 1(1), 43-48. https://doi.org/10.31838/ESA/01.01.08
9. James, A., Thomas, W., & Samuel, B. (2025). IoT-enabled smart healthcare systems: Improvements to remote patient monitoring and diagnostics. Journal of Wireless Sensor Networks and IoT, 2(2), 11-19.
10. Carlos, A., José, D., & Antonio, J. A. (2025). Structural health monitoring and impact in civil engineering. Innovative Reviews in Engineering and Science, 3(1), 1–8. https://doi.org/10.31838/INES/03.01.01
11. Min, A. K., Thandar, N. H., & Htun, Z. T. (2025). Smart sensors embedded systems for environmental monitoring system integration. Journal of Integrated VLSI, Embedded and Computing Technologies, 2(3), 1–11. https://doi.org/10.31838/JIVCT/02.03.01
12. Slavin, R. E. (2009). Segmental arterial mediolysis: course, sequelae, prognosis, and pathologic–radiologic correlation. *Cardiovascular Pathology*, *18*(6), 352-360. https://doi.org/10.1016/j.carpath.2008.09.001
13. Shenouda, M., Riga, C., Naji, Y., & Renton, S. (2014). Segmental arterial mediolysis: a systematic review of 85 cases. *Annals of vascular surgery*, *28*(1), 269-277. https://doi.org/10.1016/j.avsg.2013.03.003
14. van Twist, D. J., Appelboom, Y., Magro-Checa, C., Haagmans, M., Riedl, R., Yazar, O., ... & Mostard, G. J. (2024). Differentiating between segmental arterial mediolysis and other arterial vasculopathies to establish an early diagnosis–a systematic literature review and proposal of new diagnostic criteria. *Postgraduate Medicine*, *136*(1), 1-13. https://doi.org/10.1080/00325481.2023.2288561
15. Skeik, N., Olson, S. L., Hari, G., & Pavia, M. L. (2019). Segmental arterial mediolysis (SAM): systematic review and analysis of 143 cases. *Vascular Medicine*, *24*(6), 549-563. https://doi.org/10.1177/1358863X19873410
16. Pillai, A. K., Iqbal, S. I., Liu, R. W., Rachamreddy, N., & Kalva, S. P. (2014). Segmental arterial mediolysis. *Cardiovascular and Interventional Radiology*, *37*(3), 604-612. https://doi.org/10.1007/s00270-014-0859-4
17. Kapoor, R., & Iyer, S. (2024). Renewable Energy Integration in Sustainable Healthcare Systems. *International Journal of SDG's Prospects and Breakthroughs*, *2*(4), 7-12.
18. Nymana, F. G., & Usun, S. (2025). Cross-cultural neurocognitive profiling of food cue reactivity using EEG and AI: Toward personalized interventions for maladaptive eating. Advances in Cognitive and Neural Studies, 1(1), 39–48.
19. Barhoumia, E. M., & Khan, Z. (2025). Predictive analysis of climate change impact using multiphysics simulation models. Bridge: Journal of Multidisciplinary Explorations, 1(1), 23–30.
20. Sindhu, S. (2024). A blockchain-enabled framework for secure data exchange in smart urban infrastructure. Journal of Smart Infrastructure and Environmental Sustainability, 1(1), 31–43.
21. Surendar, A. (2025). Wearable sensor analysis of biomechanics in Yoga Asanas for posture correction. Journal of Yoga, Sports, and Health Sciences, 1(2), 1–7.
22. Rahman, F., & Prabhakar, C. P. (2023). IoT-integrated precision irrigation systems for enhancing water use efficiency in semi-arid agro-ecosystems. National Journal of Smart Agriculture and Rural Innovation, 1(1).
23. Tudevdagva, U., & Shimada, T. (2023). AI-powered crop disease detection using edge computing for smallholder farming systems. National Journal of Smart Agriculture and Rural Innovation, 1(1).

24. Patel, P., & Dusi, P. (2023). Digital twin models for predictive farm management in smart agriculture. National Journal of Smart Agriculture and Rural Innovation, 1(1).
25. Lalit, D., & Chakma, S. (2023). Community-based smart agriculture hubs for rural innovation and capacity building. National Journal of Smart Agriculture and Rural Innovation, 1(1).
26. Mori, T., & Geweely, N. (2023). Integrating indigenous knowledge into forest governance: Toward culturally rooted climate adaptation strategies. National Journal of Forest Sustainability and Climate Change, 1(1).
27. Batiha, B., & El-Fahmy, A. (2023). Biodiversity corridors in fragmented forest landscapes: Enhancing connectivity for climate-resilient ecosystems. National Journal of Forest Sustainability and Climate Change, 1(1).