Review Article

Spontaneous Coronary Artery Dissection in Diabetes Mellitus: Pathophysiological Mechanisms and Clinical Implications

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ABSTRACT

Background: Spontaneous coronary artery dissection (SCAD) is a non-atherosclerotic, uncommon cause of acute coronary syndrome, which is most frequently observed in younger women and is frequently not associated with conventional cardiovascular risk factors. Diabetes mellitus is infrequently reported among patients with SCAD, with a prevalence below 5%, yet metabolic abnormalities may influence vascular integrity and clinical outcomes.

Objective: This review examines potential mechanistic links between diabetes mellitus and SCAD, outlines diagnostic and treatment implications, and research gaps that are pertinent to personalised care.

Methods: A narrative review of observational studies, registries, reviews, and case reports published up to June 2025 was conducted using PubMed, Scopus, and Web of Science.

Key findings: Available evidence indicates that endothelial dysfunction, oxidative stress, insulin resistance, and chronic hyperglycaemia act as vascular modifiers rather than direct etiological factors for SCAD. Case reports describe SCAD occurring during acute metabolic stress, including diabetic ketoacidosis. Conservative management remains the preferred strategy in clinically stable patients.

Conclusion: Even though diabetes mellitus is not a traditional risk factor of SCAD, metabolic dysregulation can affect the manifestation of the disease, vascular healing and clinical outcomes. Prospective studies and biomarker-driven approaches are needed to clarify this relationship and support personalised management strategies.

Keywords: Spontaneous coronary artery dissection; diabetes mellitus; vascular remodeling; endothelial dysfunction; fibromuscular dysplasia; vasa vasorum.

INTRODUCTION

Acute coronary syndrome (ACS) and myocardial infarction (MI) most commonly result from atherosclerotic plaque rupture; however, spontaneous coronary artery dissection (SCAD) is a different non-atherosclerotic etiology that occurs without obstructive coronary artery disease. In SCAD, a spontaneous tear occurs within the arterial wall and it differs from atherosclerotic myocardial infarction, as it predominantly affects women younger than 50 years and is associated with recurrence, fibromuscular dysplasia (FMD), stress, and pregnancy [1]. In contrast to atherosclerotic MI, SCAD is a non-atherosclerotic diagnosis that needs high-quality coronary angiography with intracoronary imaging to distinguish it with plaque rupture or damage. Although diabetes mellitus causes endothelial dysfunction, vascular smooth muscle remodelling, and arterial stiffness which may weaken arterial integrity and influence disease expression and recovery [2]. Cardiac rehabilitation is an important component of SCAD management; however no standardized treatment strategy currently exists, and management is individualized, involving careful monitoring, pharmacotherapy, and revascularization only when clinically required [3]. Diabetes is uncommon in SCAD, with a reported prevalence of approximately 3.9%. This represents an important clinical knowledge gap; as SCAD predominantly affects individuals without traditional cardiovascular disease [4]. Therefore, optimal management of diabetes may influence vascular health and modify SCAD-related outcomes [5]. SCAD is associated with hormonal factors like estrogen, progesterone, and genetic predisposition, which may increase the risk of weakening arteries [6]. This review explores the mechanistic and clinical associations between diabetes mellitus and SCAD, outlining diagnostic issues, management issues, and new precision medicine strategies. Knowledge of metabolic modifiers like diabetes can be used to narrow the risk stratification and management in SCAD.

METHODOLOGY

This study was conducted as a narrative review. Electronic searches were conducted in PubMed, Scopus, and Web of Science using the keywords "spontaneous coronary artery dissection," "diabetes mellitus," "endothelial dysfunction," and "vascular remodeling." Studies published up to June 2025 were included, comprising observational studies, registry data, narrative reviews, systematic reviews, and case reports. Studies addressing atherosclerotic coronary artery disease without reference to SCAD were excluded from the review.

Pathophysiology of spontaneous coronary artery dissection:

Spontaneous coronary artery dissection is a rare condition with incompletely understood pathogenesis [7]. SCAD is caused by the separation of the coronary artery wall, which is most often associated with intimal disruption or intramural haematoma, which causes the constriction of the true lumen and myocardial ischemia [8,9]. This process creates a false lumen within the vessel wall, although the initiating sequence of events in SCAD remains uncertain [10,11]. Changes in the vasa vasorum have been suggested to play a role in intramural haematoma formation in SCAD, but current evidence remains indirect [6]. SCAD is more prevalent in females; this could be explained by hormonal factors, sex-specific biology, stress, and underlying conditions, including fibromuscular dysplasia [12]. Hormonal influences, pregnancy-related vascular changes, and inherited connective tissue disorders contribute to arterial wall vulnerability in SCAD [13]. Physical or emotional stress has been reported as a potential trigger for SCAD, probably through a transient increase in catecholamines and arterial shear stress [14,15]. A study by Vanessa et al. observed no connection between autoimmune disease and SCAD, indicating non-autoimmune mechanisms [16]. Arterial walls are weakened by connective tissue problems such as fibromuscular dysplasia, vascular Ehlers-Danlos syndrome, and other hereditary conditions such as Marfan and Loeys-Dietz, which predispose them to SCAD [17]. High SCAD polygenic scores from everyday gene changes increase risk in family and non-family cases due to inherited patterns rather than rare genes [18]. Genomics study found rare genes associated with inherited vasculopathies like those for vascular Elhers-Danlos syndrome (COL3A1), Marfan (FBN1), Loeys-Dietz (TGFBRS), LOX/FLNA in aortic aneurysms and new genes (ADAMTSL4, LRP1) all linked to high-risk SCAD disease features [19,20].

Limitations of existing SCAD models:

Most SCAD studies involve small groups of patients lacking prospective observational studies, limiting robust evidence for optimal treatment strategies. The mechanism of disease of SCAD is more of a hypothesis than a definite clinical evidence of which is the starting event: intimal tear or intramural haematoma. Diagnosing SCAD is challenging because angiograms miss small wall bleeds and low clinical suspicion postpones imaging. The contribution of hormonal factors to SCAD development remains incompletely defined. Currently, no standardized genetic testing strategies or long-term outcome studies are available.

Table 1: Comparison of Spontaneous Coronary Artery Dissection and Atherosclerotic Acute Artery Syndrome

Feature	SCAD	Atherosclerotic acute coronary syndrome [ACS]
Primary structural event	Separation within arterial walls	Destruction of lipid rich layer
Mechanism of lumen compromise	Compression by intramural hematoma	Formation of thrombus over ruptured plaque
Composition of vessel wall	Interface of media	Initimal atherosclerotic core
Typical angiographic pattern	Double lumen appearance or diffuse smooth narrowing	Irregular stenosis with calcification
Mechanical response to PCI	High risk of spreading	Stabilize lesion
Healing tendency	Often spontaneous vessel remodeling	Require pharmacological plaque stabilisation
Recurrence behaviour	Involve different arterial segments	Progression of existing disease
Therapeutic focus	Hemodynamic stabilisation and wall stress reduction	Use of anti thrombotics and lipid lowering therapy
Role of diabetes	Possible vascular modifier, worsens healing	Major risk factor

Abbreviations: SCAD, spontaneous coronary artery dissection; ACS, acute coronary syndrome; PCI, percutaneous coronary intervention.

Diabetes as a metabolic risk modifier:

In diabetes, enhancing protective and regenerative pathways in endothelial cells, such as activating anti-inflammatory and antioxidant gene programs, may help prevent vascular complications even when glycemic control is suboptimal [21]. Extracellular vesicle and microRNA abnormal release in diabetes changes cell communication and causes diabetic vascular disease progression [22]. Hyperglycemia and insulin resistance disrupt endothelial function by increasing oxidative stress, mitochondrial dysfunction and inflammatory signaling thereby weakening vascular integrity. This impairs endothelial integrity, by reducing nitric oxide bioavailability, impairing normal vasodilation and relaxation of blood vessels [23]. Advanced Glycation End Products (AGEs) are harmful substances formed when sugar sticks to proteins and fats in the body, accumulation of AGEs can damage various tissues and extracellular matrix structures contributing to age-related diseases like diabetes and CVD [24,25]. Glycemic variability is a better predictor of vascular damage than HbA1c since glucose variability results in greater oxidative stress than sustained high glucose [25]. In diabetes, microangiopathy damages the vasa vasorum and endothelial cells, weakening the vessel wall [26]. Small coronary vessels in type 2 diabetes have been observed to have increased medial wall thickness and a decrease in lumen size because of hypertrophic remodeling . Most available evidence relates to type 2 diabetes, while data in type 1 diabetes are primarily to case reports [27].

Table 2: Potential Vascular Effects of Diabetes mellitus related to SCAD

Biological level	Metabolic disturbance	Vascular Effect	Possible influence in SCAD
Molecular	Persistent hyperglycemia	Protein cross linking and oxidative stress	Decreased arterial elasticity
Endothelial	Insulin resistance	Impaired nitric oxide signalling	Altered vascular activity
Microvascular	Diabetic microangiopathy	Dysfunction of vasa vasorum	Increased wall vulnerability
Cellular remodeling	Hypertrophy of smooth muscle	Thickening of medial wall	Reduces structural adaptability
Systemic	Variability of glycemic levels	Activation of inflammatory mediators	Delayed vascular repair

Linking diabetes to SCAD: clinical and mechanistic evidence:

Spontaneous coronary artery dissection has been reported in association with diabetic ketoacidosis in people who have diabetes [28]. Diabetic ketoacidosis contributes to myocardial injury through hyperglycaemia, toxicities, acidity, and electrolyte disturbances, leading to reduced blood flow to the coronary artery, causing irregular heartbeats and tissue damage. Diabetic ketoacidosis causes rapid production of ketone bodies due to triggers generated by stress hormones due to reduced oxygen delivery to the heart muscle. Severe metabolic stress during diabetic ketoacidosis causes myocardial ischemic injury due to ketone buildup. Several case reports describe patients presenting with new-onset diabetic ketoacidosis associated with multi-vessel SCAD. In a 19-year-old female with existing Type 1 DM, diabetic ketoacidosis was associated with STEMI; a later angiogram revealed SCAD [5]. SCAD is associated with pregnancy, hormonal status, and emotional stress, which weaken artery walls and increase the risk for the development of SCAD [29]. During pregnancy, hormones in the blood increase that includes progesterone and estrogen, which bring about hormonal changes such as premenstrual/menstrual phases on contraceptive. postmenopausal therapy, or infertility treatments [30,31.] Emotional stress causes an increase in adrenaline and blood pressure, weakening arteries often associated with fibromuscular dysplasia[4].

Diagnostic considerations in diabetic SCAD patients:

In patients with diabetes, SCAD diagnosis is further complicated by atypical ACS presentations and autonomic neuropathy, necessitating early use of intracoronary imaging . Silent ischemia and unusual symptoms such as dyspnoea and fatigue are observed in patients with diabetes mellitus and acute coronary syndrome. Cautious assessment, glucose monitoring, and supportive diagnostic measures such as ECG and biomarkers are required [32]. Cardiovascular autonomic neuropathy (CAN) is a serious complication of diabetes that is diagnosed using autonomic function tests that check heart rate and BP response [33]. Patients with type 2 diabetes mellitus presenting with acute coronary syndrome show higher thrombus burden and greater microvascular dysfunction compared to non-diabetics [34]. Diabetic STEMI patients show more severe coronary lesions and more multi vessel disease, indicating the impact of glycemic dysregulation on atherosclerosis and plaque burden [35]. SCAD is classified on coronary angiography by the SAW system: Type 1 shows a classic double lumen with contrast staining, Type 2 shows long diffuse arrowing (>20 mm), Type 3 shows short stenosis, Type 4 shows distal total occlusion, and an intermediate type 1/2 combines features of types 1 and 2 [36]. Optical coherence tomography (OCT) is important in the diagnosis of SCAD as it offers high-resolution images of the coronary arterial wall and also direct visualisation of intramural haematoma and initial tears and false lumen formation [37]. Intravenous ultrasound (IVUS) was used to confirm the diagnosis of SCAD; it can help in guiding the stent sizing and implantation, assist in procedural planning, and contribute to recovery of left ventricular function [38]. Coronary vasospasm can contribute to SCAD by causing constriction of the coronary artery and by triggering or worsening the arterial tear and formation of a false lumen [39]. The diagnosis of SCAD in diabetic patients is uniquely complex due to coexisting conditions, and angiography is often biased towards atherosclerosis, necessitating advanced imaging to ensure correct diagnosis and management.

Management and therapeutic implications:

The SCAD management is largely founded on proper diagnosis, symptom management, outcome management, and recurrence prevention, which is specialised to the individual patient. No prospective randomised data is present that can evaluate different drug therapies for the management of SCAD [41]. SCAD is preferentially managed with conservative therapy and optimal medical therapy due to its higher recovery rates rather than percutaneous coronary artery intervention in clinically stable patients, as it is associated with more risk and recurrence [42]. However, percutaneous coronary artery intervention is considered in higher-risk cases that include ischemia, left main/proximal artery involvement, or instability, requiring careful weighing of risks and benefits [9]. Coronary artery bypass grafting is considered in the failure of PCI in SCAD patients [41]. In SCAD, antithrombotic agents and also P2Y12 inhibitors are avoided as they cause haematoma growth; GPIIb/IIIa blockers should be avoided and anticoagulant is generally discontinued once SCAD is confirmed, unless another clear clinical indication exists [43]. Dual anti-platelet therapy may be considered, typically aspirin with clopidogrel, depending on clinical context [44]. Beta blockers are used in hypertension to reduce the load on the heart, and for reducing vessel wall stress in SCAD, thrombolysis is contraindicated [45]. Add antianginals such as calcium channel blockers, nitrates, and ranolazine for symptomatic relief [43]. Use angiotensin-converting

enzyme inhibitors, angiotensin II receptor blockers, or mineralocorticoid receptor agonists if left ventricular dysfunction develops. Regular statin treatment is not recommended in patients with SCAD unless it is necessary in the management of cardiovascular risks. However, long-term management of SCAD mainly focuses on fibromuscular dysplasia, monitoring for chest pain and recurrence, and participation in heart health programs [46]. Diabetes may influence healing and management complexity (up to 35% in planned PCI), causing delays in healing and raising risks. Tailor therapy, prioritising conservative management for stable diabetic SCAD [47]. Good glycemic control is associated with vascular recovery by reducing the inflammation, oxidative stress, and endothelial dysfunction in diabetic patients [48]. Poor glycemic control (high HbA1c) causes impairment in healing and increases the risk of stenosis and stroke [49]. Cardiac rehabilitation programs are group programs that encourage healthy lifestyles, risk management, symptom management, improved functioning, and reduced events to enhance quality of life [50]. Patients who have diabetes are at higher risk of cardiovascular events; hence, cardiac rehabilitation programs are proven to increase the quality of life and risk management in these patients [51]. Long-term management also includes management of stress, blood pressure and glycemic control [52]. The role of anti-inflammatory drugs in the prevention of SCAD recurrence is unclear and requires further research[9].

Precision medicine and risk stratification:

Although SCAD typically occurs in individuals without traditional risk factors, metabolic conditions such as diabetes may influence disease expression and recovery [53]. SCAD is a heterogeneous disease; a new multiomics strategy, such as genomic and other molecular profiling, has the potential to discover subtypes of SCAD, enhancing more precise risk management [54]. Cardiovascular biomarkers like cardiac troponin, CK-MB, H-FABP, BNP/NT-pro BNP, ST2, and galectin-3 play an important role for diagnosis, prognosis, and personalised management of cardiovascular diseases [53]. In diabetes, precision medicine can use genetic information to identify patients with vascular or metabolic risk, helping to predict SCAD and other vascular complications [55]. Integrating polygenic risk score (PRS) with metabolic markers can improve SCAD risk prediction, enabling more personalised assessment and targeted management [54]. NT-pro BNP is the strongest predictor of cardiovascular stress, where higher levels indicate a higher risk of heart disease in people with type 2 diabetes [56]. GDF-15 is a marker of blood vessel damage, showing ongoing stress and changes in the vessel walls that increase cardiovascular risk in people with diabetes [57]. Artificial intelligence (AI) facilitates interpretation of complex imaging easier to understand, helping doctors to manage difficult heart vessel anatomy during PCI [9]. Integration of coronary computed tomography angiography (CCTA) with machine learning (ML) helps to differentiate atherosclerotic plaque from coronary artery dissection, enhancing more accurate diagnosis and better guided coronary intervention [55]. Precision medicine combines molecular, imaging, and clinical data that help to identify SCAD risk in people with diabetes, allowing earlier detection and personalised care [9].

Table 3: Diagnostic and risk assessment tools in SCAD

Tool or Marker	Application
Polygenic susceptibility profiling	Identification of inherited vascular vulnerability
Natriuretic peptides and stress response markers	Prognostic evaluation
Machine learning and image interpretation	Diagnostic support
High resolution lumen and wall imaging	Structural confirmation
Metabolic and genomic evaluation	Individualised risk assessment

Research gaps and future directions:

The current literature does not contain any large-scale prospective cohort studies to study SCAD in diabetic patients. This limits the understanding of diabetes as a trigger factor for the progression of SCAD; however, it is related to cardiovascular risk in metabolic disorders. Registries and the conduct of prospective studies can fill this knowledge gap. Mechanistic and translational models in SCAD are required for uncovering disease mechanisms—with the help of animal models, cell studies, or computer models for explaining blood vessel tears from “inside out” or “outside in” processes, including trigger factors like hormones and emotional stress—and applying these findings in the proper management of SCAD rather than depending on case reports, etc. No proven blood tests or metabolic markers exist for SCAD, making early diagnosis and risk prediction difficult, Particularly in patients with diabetes. Recent studies mainly focus on diagnostic scans like OCT for spotting vessel tears, but metabolic biomarkers need prospective cohort studies. This restricts the creation of precision medicine strategies and pathophysiology of SCAD. This gap can be filled by future research on metabolomics based on patient registries. The timing and predictors of SCAD recurrence remain unpredictable; it is often linked to high blood pressure or fibromuscular dysplasia, however reliable predictive tools for identifying high-risk individuals remain limited, so management therefore primarily relies on blood pressure control, particularly with beta-blockers and close clinical surveillance, rather than finding newer treatments. Better prediction is required with respect to diabetes comorbidity using prospective cohorts and biomarkers.

CONCLUSION

Spontaneous coronary artery dissection and diabetes mellitus is a complicated clinical phenomenon that is not similar to atherosclerotic acute coronary syndromes. Although diabetes is not a classic risk factor for SCAD, its presence may influence disease expression by promoting endothelial dysfunction, vascular inflammation, and structural weakness of the arterial wall. Current evidence

supports a conservative treatment approach in most stable patients, as revascularization carries a higher risk of procedural complications and does not consistently improve outcomes. Although the precise relationship between diabetes and SCAD remains incompletely understood, metabolic dysregulation appears to influence disease expression and recovery. Further research on metabolic and vascular biomarkers with the help of properly designed registries is required to understand this association and implement more individualized management approaches in patients with SCAD.

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