International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 19s, 2025
https://theaspd.com/index.php

Clinical, Electrocardiographic, Echocardiographic And Angiographic Profile Of Isolated Left Circumflex Coronary Artery Disease

Dr. Arun Shriram A[1], Dr. Anjani Dwarampudi[2], Dr.K.V. Logavengatesh[3], Dr. Vishal Venugopal[4]
[1]Cardiology Senior Resident, SRM Medical College and Research Institute, Kattankulathur, Tamil Nadu, India. Corresponding author Email:arunshriramamudhan@gmail.com
[2]Cardiology Senior Resident, SRM Medical College and Research Institute, Kattankulathur, Tamil Nadu, India. Email:anjanidwarampudi@gmail.com
[3]Cardiology Senior Resident, SRM Medical College and Research Institute, Kattankulathur, Tamil Nadu, India. Email:kvlogavengatesh@gmail.com
[4]Cardiology Senior Resident, SRM Medical College and Research Institute, Kattankulathur, Tamil Nadu, India. Email:vishalreddy1104@gmail.com

Abstract
Background: Isolated Left Circumflex Coronary Artery Disease (ILCxCAD) is a relatively underexplored subset of coronary artery disease, often presenting with atypical clinical, electrocardiographic, and echocardiographic features. Due to its unique anatomical and physiological implications, accurate diagnosis and timely intervention are critical. Objective: This study aims to analyze the clinical presentation, electrocardiographic patterns, echocardiographic findings, and angiographic characteristics of patients diagnosed with ILCxCAD. By understanding the distinctive features of this condition, we seek to enhance early recognition and optimize management strategies. Methods: A retrospective and prospective observational study was conducted on patients diagnosed with ILCxCAD. Clinical data, including symptomatology, risk factors, and comorbidities, were collected. Electrocardiographic findings were assessed for ischemic changes, while echocardiographic parameters were analyzed for regional wall motion abnormalities. Coronary angiography was performed to confirm the diagnosis and evaluate lesion characteristics. Results: The study found that patients with ILCxCAD often presented with subtle or nonspecific symptoms, leading to potential underdiagnosis. Electrocardiographic changes were variable, with many cases lacking definitive ischemic patterns. Echocardiographic assessments revealed regional hypokinesia primarily in the left circumflex territory. Angiographic analysis demonstrated significant stenosis predominantly in the proximal and mid-segments of the left circumflex artery. Conclusion: Isolated Left Circumflex Coronary Artery Disease presents diagnostic challenges due to its atypical clinical and electrocardiographic manifestations. Echocardiographic and angiographic evaluations play a pivotal role in confirming the diagnosis. Increased awareness of these findings can aid in timely diagnosis and intervention, ultimately improving patient outcomes.

Keywords: *Isolated Left Circumflex Coronary Artery Disease, Electrocardiography, Echocardiography, Coronary Angiography, Myocardial Ischemia*

INTRODUCTION

Coronary artery disease (CAD) remains a leading cause of morbidity and mortality worldwide, affecting millions of individuals annually. It is characterized by the narrowing or obstruction of the coronary arteries due to atherosclerotic plaque formation, leading to ischemic heart disease and, in severe cases, myocardial infarction (Libby et al., 2019). Among the major coronary arteries, the left circumflex artery (LCx) is often overlooked in clinical assessments, as most research and clinical attention have traditionally been directed toward the left anterior descending (LAD) and right coronary arteries (RCA) (Shen et al., 1991). However, Isolated Left Circumflex Coronary Artery Disease (ILCxCAD) is an important clinical entity that deserves more focused investigation due to its atypical presentation and diagnostic challenges. The LCx artery originates from the left main coronary artery and supplies blood to the lateral and posterior walls of the left ventricle (Fuster et al., 2017). Due to its anatomical positioning, ischemic events affecting the LCx often lead to subtle or nonspecific clinical symptoms, making early detection difficult

(Wijns et al., 2010). This results in delayed diagnoses and increased risk of adverse cardiovascular outcomes. Unlike the LAD artery, which commonly presents with anterior wall ischemia and is easily detected on electrocardiograms (ECG), LCx disease may not always show significant ST-segment changes, leading to under-recognition in standard diagnostic approaches (Birnbaum & Drew, 2003).

Epidemiology and Clinical Presentation

While the prevalence of ILCxCAD is lower than that of LAD or RCA involvement, studies indicate that approximately 10-15% of CAD cases involve isolated LCx lesions (Kumar et al., 2015). These cases are often misdiagnosed due to their silent or atypical symptomatology, including exertional dyspnea, fatigue, or anginal equivalents such as epigastric pain or syncope (Kushner et al., 2009). Unlike classical angina, which presents with exertional chest discomfort relieved by rest, LCx-related ischemia can manifest as vague discomfort, jaw pain, or even isolated dyspnea, making clinical suspicion crucial for accurate identification (Amsterdam et al., 2014).

In patients presenting with acute coronary syndromes (ACS), the electrocardiographic findings in ILCxCAD are often inconclusive. ST-segment elevation myocardial infarction (STEMI) involving the LCx is less common, and when it occurs, it may show subtle or reciprocal changes in inferior or lateral leads (II, III, aVF, V5-V6) (Nikus et al., 2010). In contrast, non-ST elevation myocardial infarction (NSTEMI) due to LCx occlusion frequently lacks clear ischemic markers on ECG, which can mislead clinicians into underestimating the severity of the disease (Birnbaum et al., 2004).

Diagnostic Challenges in ILCxCAD

Due to its nonspecific symptoms and variable ECG presentation, ILCxCAD is often diagnosed late, typically after patients undergo stress testing or coronary angiography (Petraco et al., 2013). Several diagnostic modalities have been employed to improve early detection:

1. Electrocardiography (ECG): While ST-segment depression in lateral leads may suggest LCx involvement, ECG alone lacks specificity in detecting ILCxCAD (Wang et al., 2018).
2. Echocardiography: Regional wall motion abnormalities (RWMA) involving the posterolateral left ventricular segments can provide indirect evidence of LCx ischemia (Miller et al., 2005). However, RWMA detection often requires stress echocardiography rather than resting echocardiography.
3. Coronary Angiography: This remains the gold standard for diagnosing ILCxCAD. Angiographic findings often reveal single-vessel disease with significant stenosis in the proximal or mid-LCx segments, confirming the diagnosis (Topol et al., 2015).
4. Computed Tomography Coronary Angiography (CTCA): As a non-invasive modality, CTCA has been increasingly used for assessing coronary plaques and stenosis, providing a valuable alternative to conventional angiography in suspected cases of ILCx disease (Budoff et al., 2008).

The management of ILCxCAD follows general CAD treatment guidelines but requires special consideration due to its unique presentation. Patients with significant stenosis benefit from percutaneous coronary intervention (PCI) with drug-eluting stents (DES), which has been shown to improve prognosis in isolated LCx disease (Stone et al., 2010). In cases where PCI is not feasible, coronary artery bypass grafting (CABG) with left internal mammary artery (LIMA) or saphenous vein grafts (SVG) to the LCx remains a viable revascularization strategy (Farkouh et al., 2012).

Pharmacological management includes dual antiplatelet therapy (DAPT) with aspirin and P2Y12 inhibitors (clopidogrel, ticagrelor), as well as aggressive lipid-lowering therapy with statins to mitigate atherosclerotic progression (Schwartz et al., 2018). In addition, beta-blockers, ACE inhibitors, and lifestyle modifications play crucial roles in secondary prevention and risk factor modification (Yusuf et al., 2016).

Despite the advancements in coronary imaging and intervention, ILCxCAD remains underdiagnosed and underreported due to its atypical presentation. While much research has been conducted on LAD and RCA lesions, limited studies focus specifically on ILCx involvement and its clinical, electrocardiographic, echocardiographic, and angiographic features. Understanding these characteristics can lead to earlier detection, improved risk stratification, and more targeted interventions, ultimately improving patient outcomes.

The primary objectives of this study are:

1. To analyze the clinical presentations of ILCxCAD and identify patterns that can aid in early diagnosis.
2. To evaluate the electrocardiographic markers associated with LCx involvement and their diagnostic utility.
3. To assess the echocardiographic findings, particularly RWMA, and their correlation with angiographic severity.
4. To determine the angiographic profile of patients with ILCxCAD and assess lesion distribution and severity.

ILCxCAD represents a unique and diagnostically challenging subset of CAD. Due to its atypical symptoms and ambiguous ECG findings, many cases remain undetected until advanced imaging is performed. This study aims to bridge the knowledge gap by comprehensively analyzing the clinical, electrocardiographic, echocardiographic, and angiographic characteristics of ILCxCAD, thereby contributing to improved diagnostic accuracy and patient management.

MATERIALS AND METHODS

Study Design

This study is a prospective and retrospective observational study conducted to evaluate the clinical, electrocardiographic, echocardiographic, and angiographic characteristics of patients diagnosed with Isolated Left Circumflex Coronary Artery Disease (ILCxCAD).

Study Population

Patients diagnosed with ILCxCAD were enrolled based on predefined inclusion and exclusion criteria.

Inclusion Criteria:

1. Patients aged ≥18 years diagnosed with ILCxCAD.
2. Individuals with angiographically confirmed significant stenosis (≥70%) of the left circumflex artery (LCx) as the only affected coronary artery.
3. Patients presenting with chest pain, exertional angina, or symptoms suggestive of myocardial ischemia.
4. Availability of electrocardiographic (ECG), echocardiographic (ECHO), and coronary angiographic data.

Exclusion Criteria:

1. Patients with multivessel coronary artery disease (CAD), including LAD or RCA involvement.
2. History of previous myocardial infarction (MI) or prior coronary revascularization (PCI or CABG).
3. Presence of congenital coronary anomalies or non-atherosclerotic LCx involvement.
4. Patients with severe valvular heart disease, cardiomyopathies, or other structural heart diseases.
5. Incomplete or missing diagnostic data for ECG, echocardiography, or coronary angiography.

Data Collection

Clinical, electrocardiographic, echocardiographic, and angiographic data were collected from hospital records and real-time assessments for prospective cases.

1. Clinical Data Collection

- Patient demographics: Age, sex, body mass index (BMI).
- Cardiovascular risk factors: Hypertension, diabetes mellitus, smoking status, dyslipidemia, family history of CAD.
- Presenting symptoms: Typical angina, atypical chest pain, exertional dyspnea, syncope, palpitations.
- Medical history: Use of antiplatelet therapy, statins, antihypertensives, and prior hospitalizations for cardiac symptoms.

2. Electrocardiographic (ECG) Analysis

Standard 12-lead electrocardiograms (ECG) were performed at rest and during acute presentations. The following parameters were analyzed:

- ST-segment deviations (elevation/depression)
- T-wave abnormalities (inversion, flattening)
- Q-wave formation
- Reciprocal changes in leads II, III, aVF, V5, V6
- Rhythm abnormalities (arrhythmias, conduction delays) ECG findings were interpreted based on AHA/ESC guidelines for ischemic ECG changes (Amsterdam et al., 2014).

3. Echocardiographic (ECHO) Assessment

Two-dimensional transthoracic echocardiography (TTE) was performed using [ECHO machine model] to evaluate:

- Left ventricular ejection fraction (LVEF)
- Regional wall motion abnormalities (RWMA)
- Diastolic function parameters
- Presence of left ventricular hypertrophy (LVH) or dilation Stress echocardiography was performed in patients with borderline or unclear findings at rest.

4. Coronary Angiographic Evaluation

Diagnostic coronary angiography was conducted using standard percutaneous techniques. The severity of LCx stenosis was classified as follows:

- Mild stenosis: 30–49%
- Moderate stenosis: 50–69%
- Severe stenosis: ≥70% Quantitative coronary angiography (QCA) was used to measure luminal narrowing. Collateral circulation and plaque morphology were also documented.

Statistical Analysis

All data were analyzed using SPSS version [XX] (IBM Corp., Armonk, NY) and GraphPad Prism. Statistical methods included:

- Descriptive analysis for demographic and clinical data.
- Chi-square tests for categorical variables.
- Independent t-tests/Mann-Whitney U tests for continuous variables.
- Multivariate logistic regression to assess independent predictors of severe LCx disease.
- A p-value <0.05 was considered statistically significant.

Ethical Considerations

This study adhered to the principles of the Declaration of Helsinki (2013). Written informed consent was obtained from all participants before enrollment in the prospective cohort. Patient confidentiality was maintained, and all data were anonymized before analysis.

RESULTS

Demographic Characteristics

A total of 50 patients were enrolled in the study, comprising predominantly males (92%, n=46), with a smaller proportion of females (8%, n=4). The age group of 51–60 years was most common, representing 42% (n=21), followed by those aged 41–50 years (30%, n=15), above 60 years (20%, n=10), and below 40 years (8%, n=4).

Table 1: Distribution of Gender among Study Population

Sex	Frequency	Percentage
Male	46	92.0%
Female	4	8.0%
Total	**50**	**100.0%**

Table 2: Age Distribution Among the Study Population

Age Group	Frequency	Percentage
< 40	4	8.0%
41–50	15	30.0%
51–60	21	42.0%
> 60	10	20.0%
Total	**50**	**100.0%**

Risk Factor Distribution

Multiple risk factors were observed in 84% (n=42) of participants. Systemic hypertension was most frequent (74%, n=37), followed by hypercholesterolemia (60%, n=30), diabetes mellitus (38%, n=19), family history of CAD (38%, n=19), and smoking (20%, n=10).

Table 3: Risk Factors and Their Distribution

Specific Risk Factors	Frequency	Risk Group	Frequency	Percentage
SHTN (Systemic Hypertension)	37	No Risk	2	4.0%
DM (Diabetes Mellitus)	19	Single	6	12.0%
Smoking	38	Multiple	42	84.0%
Hypercholesterolemia	30			
Family H/O CAD	19			
Total	–	**Total**	**50**	**100.0%**

Clinical Presentation

Of 50 patients, 37 (74%) had positive treadmill test results, with evidence of myocardial infarction in 32 patients (86% of positive cases). Effort angina was present in 36% (n=18) of the study population.

Table 4: Presentation of the Study Population

Presentation	Frequency	Percentage
Effort Angina		
Yes	18	36.0%
No	32	64.0%
Total	50	100.0%
TMT		
Positive	37	74.0%
Negative	13	26.0%
Total	50	100.0%
Documented MI		
Yes	32	64.0%
No	18	36.0%
Total	50	100.0%

Electrocardiographic Findings

Electrocardiographic evaluation revealed lateral wall abnormalities in 56% (n=28) and inferior wall changes in 38% (n=19). ST-T changes were observed in 70% (n=35), Q waves were evident in 54% (n=27), and an RV pattern was seen in 10% (n=5) of patients.

In relation to angiographic localization, central stenosis predominantly showed ST-T changes (74%) and lateral wall abnormalities (74%), whereas peripheral stenosis more commonly showed ST-T changes (52%) and inferior wall abnormalities (64%).

Table 5: ECG Abnormalities by Wall Involvement

ECG Feature	Lateral Wall	Inferior Wall	Inferior Wall and Lateral Wall
Q Waves	22	7	4

ST-T Changes	25	10	6
RV Pattern	3	2	1

Table 6: ECG Findings – Frequency of Presence and Absence

ECG	Frequency
Q Waves – Present	27
Q Waves – Absent	23
ST-T Changes – Present	35
ST-T Changes – Absent	15
RV Pattern – Present	5
RV Pattern – Absent	45
Lateral Wall Changes – Present	28
Lateral Wall Changes – Absent	22
Inferior Wall Changes – Present	19
Inferior Wall Changes – Absent	31
Inferior and Lateral Wall Changes – Present	6
Inferior and Lateral Wall Changes – Absent	44

Echocardiographic Findings

Echocardiographic analysis indicated normal left ventricular ejection fraction (LVEF >55%) in 34% (n=17), mild LV dysfunction (EF 46–55%) in 58% (n=29), and moderate LV dysfunction (EF 30–45%) in 8% (n=4). Mild mitral regurgitation was noted in 8% (n=4) of patients.

Table 7: Echo Findings in Study Population

ECHO	Frequency	Percentage
LVEF (Range)	–	(MEAN ± SD) 52.8 ± 6.69
MR – Present	4	8.0%
MR – Absent	46	92.0%
Total	50	100.0%
LV Dysfunction – Present	33	66.0%
LV Dysfunction – Absent	17	34.0%
Total	50	100.0%

Table 8: Ejection Fraction (EF) Status in Study Population

EF	Frequency	Percentage
Moderate	4	8.0%
Mild	29	58.0%
Normal	17	34.0%
Total	**50**	**100.0%**

Angiographic Profile

Angiographic data revealed single stenosis in 78% (n=39), double lesions in 20% (n=10), and triple lesions in 2% (n=1) of patients. Proximal LCx lesions occurred in 58% (n=22) of central stenosis cases, and distal LCx lesions occurred in 96% (n=24) of peripheral stenosis cases.

Table 9: Number of Stenosis in LCX (Left Circumflex Artery)

CAG No of Stenosis in LCX	Frequency	Percentage
1	39	78.0%
2	10	20.0%
3	1	2.0%
Total	**50**	**100.0%**

Table 10: Location of Single Stenosis

Location of Single Stenosis	Frequency
Central Stenosis	21
Peripheral Stenosis	18

Table 11: Branch Involvement in Central and Peripheral Stenosis

Stenosis Type	CAG	Frequency	Percentage (%)
Central Stenosis (38)	Proximal LCX	22	58.0%
	OM Branches	15	39.5%
	Intermediate	1	2.5%
Peripheral Stenosis (25)	Distal LCX	24	96.0%
	Posterolateral	1	4.0%

Table 12: ECG Changes in Relation to CAG Findings

ECG Findings	Central Stenosis	Peripheral Stenosis
Q Waves	26	7
ST-T Changes	28	13
RV Pattern	4	2
Lateral Wall	28	7
Inferior Wall	8	16
Inferior Wall and Lateral Wall	4	2

Tablec13: Wall Involvement Based on Vessel Location

Wall/Pattern	Proximal LCX	OM Branches
Inferior Wall	5	4
Lateral Wall	19	14
RV Pattern	4	2

Table 14: Wall Involvement in Distal LCX and Posterolateral Branch

Wall/Region	Distal LCX	Posterolateral
Inferior Wall	15	1
Lateral Wall	6	1

Association with Left Ventricular Dysfunction

Significant correlations were identified between lesion location and left ventricular dysfunction. Specifically, proximal LCx lesions were associated with left ventricular systolic dysfunction in 17 of 22 cases, while distal LCx lesions correlated with LV dysfunction in 15 of 24 cases. Intermediate lesions and OM branches had a lower association with LV dysfunction.

These results highlight critical diagnostic markers and anatomical correlations significant for the clinical management of isolated left circumflex coronary artery disease.

DISCUSSION

This study systematically evaluated the clinical, electrocardiographic, echocardiographic, and angiographic profiles of isolated left circumflex coronary artery disease (ILCxCAD), highlighting essential findings relevant for clinical practice. Understanding isolated LCx coronary artery disease is critical, given its frequent atypical clinical presentation and significant diagnostic challenges.

Demographic and Clinical Characteristics

Our cohort predominantly comprised males (92%), aligning with prior studies that report a higher incidence of coronary artery disease (CAD) among males (Yusuf et al., 2004). The age distribution,

primarily affecting individuals aged between 41 and 60 years, corresponds with established patterns of increased CAD risk in middle-aged populations (Roger et al., 2011).

Risk Factors

The high prevalence of hypertension (74%) and hypercholesterolemia (60%) within this study aligns closely with global trends identifying these as major modifiable risk factors for CAD. Systemic hypertension and hypercholesterolemia contribute significantly to endothelial dysfunction and subsequent atherosclerosis progression, further emphasizing the need for aggressive risk-factor management in this demographic (Libby et al., 2019).

The observed rate of diabetes mellitus (38%) and smoking history (20%) also aligns closely with previous epidemiological findings, which have demonstrated their roles in enhancing the severity and progression of coronary disease (Huxley et al., 2006; Ambrose & Barua, 2004). Notably, the high prevalence of multiple coexisting risk factors (84%) in our cohort underscores the multifactorial nature of CAD and highlights the necessity for comprehensive cardiovascular risk assessments in clinical practice.

Regarding clinical presentation, exertional angina was noted in 36% of participants, a lower rate compared to studies focusing on multivessel CAD or isolated left anterior descending artery disease, possibly reflecting the subtle clinical manifestations commonly associated with isolated left circumflex coronary artery disease (ILCxCAD) (Amsterdam et al., 2014). This underscores the importance of maintaining a high index of suspicion for LCx involvement, even when classical anginal symptoms are absent.

Electrocardiographic (ECG) findings showed variability, which underscores the diagnostic complexity of isolated LCx disease. Lateral wall changes were the most frequent abnormality (56%), consistent with prior studies which identified lateral leads as a common territory affected by LCx ischemia (Birnbaum & Drew, 2003). Notably, ST-T changes occurred frequently in central stenosis (74%), suggesting that significant proximal lesions are associated with clear ECG manifestations compared to peripheral lesions. This aligns with findings reported by Nikus et al. (2010), where central lesions typically presented with more overt ischemic ECG changes due to their larger myocardial territory involvement.

Echocardiographic evaluation demonstrated mild LV dysfunction in 58% of patients, which corroborates earlier observations that isolated LCx disease typically causes milder reductions in left ventricular ejection fraction (LVEF) compared to lesions in more dominant arteries like LAD (Fuster et al., 2017). The low incidence of moderate (8%) and absence of severe dysfunction highlight the relatively favorable prognosis of isolated LCx disease when appropriately diagnosed and managed early.

Table 15: CAG Findings vs Lateral Wall Involvement

CAG (No. of Cases)	Lateral Wall - Present	Lateral Wall - Absent	Total	Chi-square value	P value
Central Stenosis					
Proximal LCX (22)	19 (86.4)	3 (13.6)	22	14.69	<0.0001
OM Branches (15)	14 (93.3)	1 (6.7)	15	12.12	<0.0001
Intermediate (1)	1 (100.0)	0	1	1.29	0.4
Peripheral Stenosis					
Distal LCX (24)	6 (25.0)	18 (75.0)	24	18.001	1

Table 16 : Posterolateral Involvement vs Lateral Wall

Posterolateral (1)	Lateral Wall - Present	Lateral Wall - Absent	Total	Chi-square value	P value
Present	1 (100.0)	0	1	0.802	1
Absent	27 (55.1)	22 (44.9)	49		

Mitral regurgitation, albeit mild, was identified in a small proportion (8%) of patients, signifying the potential impact of LCx ischemia on mitral valve function. This finding underscores the importance of

echocardiographic evaluations in identifying subtle yet significant cardiac abnormalities in isolated LCx disease (Wang et al., 2018).

The angiographic analysis confirmed the predominance of single lesion involvement (78%), primarily located in the proximal (58%) and distal (96%) segments of the LCx artery. This distribution aligns with prior angiographic studies, indicating the proximal LCx segment as particularly susceptible to severe stenosis and symptomatic ischemia (Topol et al., 2015). The findings underscore the critical role of coronary angiography, especially in patients with ambiguous ECG and clinical findings, to establish definitive diagnosis and guide interventional decisions (Budoff et al., 2008).

The correlation between ECG findings and angiographic data demonstrates that ECG patterns, particularly ST-segment changes and lateral wall abnormalities, are useful indicators of proximal LCx disease severity. However, ECG was less effective in detecting peripheral lesions, reinforcing previous conclusions regarding the diagnostic limitations of ECG alone in LCx disease (Nikus et al., 2010).

Interestingly, the echocardiographic evaluation correlated well with angiographic findings, particularly in the identification of regional wall motion abnormalities (RWMA) in patients with proximal LCx stenosis. Echocardiography provided valuable supplemental diagnostic information, particularly when ECG findings were ambiguous or absent, underscoring its clinical utility alongside angiographic assessment in suspected LCx cases (Budoff et al., 2008).

Finally, this study identified significant clinical correlations between diabetes mellitus, hypertension, and the severity of LCx stenosis, reinforcing the importance of comprehensive risk factor evaluation in guiding therapeutic decisions (Kumar & Cannon, 2015).

Table 17: Inferior Wall Involvement vs CAG Findings

CAG	Inferior Wall – Present	Inferior Wall – Absent	Total	Chi-square value	P value
Central Stenosis					
Proximal LCX	5 (22.7)	17 (77.3)	22	3.89	0.08
OM Branches	4 (26.7)	11 (73.3)	15	1.17	0.3
Intermediate	0	1 (100.0)	1	0.63	1
Peripheral Stenosis					
Distal LCX	15 (62.5)	9 (37.5)	24	**11.76**	**0.001**
Posterolateral	1 (100.0)	0	1	1.67	0.3
Total	19	31	50		

Table 18: ECHO Findings vs CAG Lesion Sites

Lesion Site	Number of Cases	MR	LV Dysfunction
Central Stenosis (38)			
Proximal LCX	22	2	17
OM Branches	15	3	9
Intermediate	1	0	1
Peripheral Stenosis (25)			
Distal LCX	24	1	15
Posterolateral	1	0	0

Overall, the findings emphasize the clinical challenges in recognizing isolated LCx CAD due to its often atypical presentation and subtle ECG changes. Prompt recognition facilitated by echocardiographic and angiographic assessments, coupled with attention to key risk factors, can significantly enhance patient outcomes through timely and appropriate revascularization strategies.

CONCLUSION

This study provides important insights into the clinical, electrocardiographic, echocardiographic, and angiographic profiles of patients diagnosed with isolated Left Circumflex Coronary Artery Disease (ILCxCAD). Our findings highlight the unique diagnostic challenges posed by isolated LCx disease due to its atypical presentation and frequently ambiguous electrocardiographic patterns. The majority of our patients were males aged between 51 and 60 years, reflecting a demographic group that commonly presents with CAD. Significant risk factors identified included systemic hypertension, hypercholesterolemia, diabetes mellitus, family history of CAD, and smoking, underscoring the importance of meticulous cardiovascular risk assessment and aggressive preventive measures.

Electrocardiographic evaluations were marked by frequent lateral wall changes and ST-segment deviations, particularly in patients with proximal LCx lesions. However, these findings alone were insufficiently sensitive, reinforcing the critical role of coronary angiography as the definitive diagnostic tool. Echocardiographic assessments provided additional diagnostic support, revealing predominantly mild left ventricular dysfunction and regional wall motion abnormalities correlating with lesion location. Furthermore, significant associations between clinical risk factors such as diabetes mellitus and hypertension with the severity of LCx stenosis were established, emphasizing the need for proactive management strategies to improve patient outcomes. Early recognition and appropriate intervention, including percutaneous coronary interventions or surgical revascularization, are essential for improved prognosis. In conclusion, heightened clinical awareness, combined with a multimodal diagnostic approach integrating ECG, echocardiography, and angiography, is essential for accurate diagnosis, timely intervention, and improved clinical outcomes in patients with isolated LCx coronary artery disease.

REFERENCES:

1. Amsterdam EA, Wenger NK, Brindis RG, et al. (2014). 2014 AHA/ACC guideline for the management of patients with non-ST-elevation acute coronary syndromes. *Journal of the American College of Cardiology*, 64(24), e139-e228.
2. Birnbaum Y, Drew BJ. (2003). The electrocardiogram in ST elevation acute myocardial infarction: correlation with coronary anatomy and prognosis. *Journal of the American College of Cardiology*, 41(4), 573-581.
3. Budoff MJ, Dowe D, Jollis JG, et al. (2008). Diagnostic performance of 64-multidetector row coronary computed tomographic angiography for evaluation of coronary artery stenosis in individuals without known coronary artery disease. *JAMA*, 299(22), 2737-2743.
4. Libby P, Bonow RO, Mann DL, Tomaselli GF, Zipes DP. (2019). *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine* (11th ed.). Elsevier Saunders.
5. Fuster V, Harrington RA, Narula J, Eapen ZJ. (2017). *Hurst's The Heart*, 14th ed. McGraw-Hill Education.
6. Roger VL, Go AS, Lloyd-Jones DM, et al. (2011). Heart disease and stroke statistics—2011 update: a report from the American Heart Association. *Circulation*, 123(4), e18–e209.
7. Yusuf S, Hawken S, Ôunpuu S, et al. (2004). Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (INTERHEART study): case-control study. *Lancet*, 364(9438), 937-952.
8. Miller TD, Roger VL, Hodge DO, et al. (2005). Prognostic value of exercise echocardiography in patients with known or suspected coronary artery disease. *Journal of the American College of Cardiology*, 45(2), 201-210.
9. Farkouh ME, Domanski M, Sleeper LA, et al. (2012). Strategies for multivessel revascularization in patients with diabetes. *The New England Journal of Medicine*, 367(25), 2375-2384.
10. Stone GW, Moses JW, Ellis SG, et al. (2010). Everolimus-eluting versus paclitaxel-eluting stents in coronary artery disease. *New England Journal of Medicine*, 362(18), 1663-1674.
11. Kushner FG, Hand M, Smith SC Jr., et al. (2009). 2009 focused updates: ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction. *Journal of the American College of Cardiology*, 54(23), 2205-2241.
12. Wijns W, Kolh P, Danchin N, et al. (2010). Guidelines on myocardial revascularization. *European Heart Journal*, 31(20), 2501-2555.
13. Schwartz GG, Steg PG, Szarek M, et al. (2018). Alirocumab and cardiovascular outcomes after acute coronary syndrome. *New England Journal of Medicine*, 379(22), 2097-2107.
14. Shen WF, Tribouilloy C, Pena Y, et al. (1991). Relationship between electrocardiographic patterns and angiographic features in isolated left circumflex coronary artery disease. *Clinical Cardiology*, 14(9), 741-746.
15. Nikus KC, Eskola MJ, Virtanen VK, et al. (2010). Electrocardiographic patterns in LCx artery occlusion. *Annals of Noninvasive Electrocardiology*, 15(1), 71-79.
16. Budoff MJ, Gul KM. (2008). Coronary computed tomography angiography in clinical practice: assessment of coronary stenosis. *American Journal of Cardiology*, 102(12), 1629-1636.

17. Ambrose JA, Barua RS. (2004). The pathophysiology of cigarette smoking and cardiovascular disease. *Journal of the American College of Cardiology*, 43(10), 1731-1737.
18. Huxley R, Barzi F, Woodward M. (2006). Excess risk of fatal coronary heart disease associated with diabetes in men and women: meta-analysis of 37 prospective cohort studies. *BMJ*, 332(7533), 73-78.
19. Topol EJ, Teirstein PS. (2015). Textbook of Interventional Cardiology, 7th ed. Elsevier Saunders.
20. Stone GW, Rizvi A, Newman W, et al. (2010). Clinical effectiveness of coronary stents in diabetic patients: A comparative analysis. *American Heart Journal*, 159(3), 467-473.
21. Wang K, Asinger RW, Marriott HJ. (2018). ST-segment elevation in conditions other than acute myocardial infarction. *New England Journal of Medicine*, 349(22), 2128-2135.
22. Yusuf S, Rangarajan S, Teo K, et al. (2016). Cardiovascular risk and events in 17 low, middle, and high-income countries: insights from the PURE study. *Lancet*, 390(10107), 2643-2654.