Original Article
DOI: https://doi.org/10.70749/ijbr.v3i7.2279

INDUS JOURNAL OF BIOSCIENCE RESEARCH
https://ijbr.com.pk
ISSN: 2960-2793/ 2960-2807

Frequency of Left Ventricular Systolic Dysfunction in Patients with Wellens Syndrome Undergoing Angiography

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ARTICLE INFO

Keywords: Wellens Syndrome, Left Ventricular Systolic Dysfunction, Coronary Angiography, LAD Stenosis, Acute Coronary Syndrome.

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Declaration
Authors' Contribution
All authors equally contributed to the study and approved the final manuscript

Conflict of Interest: No conflict of interest.

Funding: No funding received by the authors.

Article History
Received: 13-06-2025 Revised: 02-07-2025
Accepted: 13-07-2025 Published: 15-07-2025

ABSTRACT

Background: Wellens syndrome is an electrocardiographic marker of critical proximal left anterior descending (LAD) artery stenosis and a strong predictor of anterior wall myocardial infarction if left untreated. **Objective:** To determine the frequency of left ventricular systolic dysfunction in patients with Wellens syndrome undergoing coronary angiography. **Methods:** This descriptive cross-sectional study was conducted at the in-patient department of the National Institute of Cardiovascular Diseases (NICVD), Karachi, Pakistan, from March 2025 to 10 June 2025. A total of 187 patients were included using non-probability consecutive sampling. Patients aged 18–60 years of both genders undergoing coronary catheterization were enrolled, excluding those with previously established LVSD. **Results:** The mean age of the study population was 51.3 ± 7.8 years, with 110 (58.8%) males and 77 (41.2%) females. Diabetes mellitus was present in 92 (49.2%) patients, hypertension in 101 (54.0%), and smoking history in 77 (41.2%). Angiography revealed proximal LAD lesions in 164 (87.7%) patients. LVSD was observed in 111 patients, giving a frequency of 59.4%. Among these, mild LVSD was present in 48 (25.7%), moderate in 39 (20.9%), and severe in 24 (12.8%). LVSD was significantly more frequent in patients with diabetes (68.5% vs. 50.0%, $p = 0.01$), hypertension (65.3% vs. 52.3%, $p = 0.04$), and triple-vessel disease (77.8%, $p < 0.001$). **Conclusion:** A high frequency of left ventricular systolic dysfunction was observed in patients with Wellens syndrome undergoing angiography, particularly in those with diabetes, hypertension, and multivessel disease.

INTRODUCTION

Left ventricular (LV) systolic dysfunction emerges as a significant concern in the clinical landscape, particularly among patients diagnosed with Wellens syndrome undergoing angiography [1]. Wellens syndrome, recognized by distinct electrocardiographic changes indicative of critical stenosis in the left anterior descending coronary artery, imposes a heightened risk on cardiac function [2]. As angiography becomes a crucial diagnostic and interventional tool for these patients, exploring the frequency of LV systolic dysfunction is imperative for comprehensive management [3]. This introductory discussion delves into the intricate relationship between Wellens syndrome and LV systolic dysfunction, shedding light on the prevalence, clinical implications, and the evolving landscape of interventions [4]. Wellens syndrome, often a precursor to anterior wall myocardial infarction, introduces a unique challenge due to its association with critical stenosis in the left anterior descending artery [5]. The frequency of LV systolic dysfunction in this specific subset of patients undergoing angiography has become a subject of increasing interest among clinicians and researchers [6]. Understanding the interplay between Wellens syndrome and LV systolic dysfunction is crucial for risk stratification and tailored therapeutic approaches [7]. Angiography, as a cornerstone in the assessment of coronary artery disease, provides clinicians with a detailed visualization of the coronary anatomy, enabling precise identification of obstructive lesions [8]. However, the impact of Wellens syndrome on LV systolic function during angiography remains a nuanced aspect requiring comprehensive exploration [9]. Recognizing the prevalence of LV systolic dysfunction in this specific clinical context is essential for refining interventional strategies and optimizing patient outcomes [10].

The hemodynamic stress induced by critical stenosis in the left anterior descending coronary artery can significantly affect LV systolic function [11]. Ischemia resulting from compromised blood flow may contribute to impaired contractility, underscoring the need to elucidate the correlation between Wellens syndrome and LV systolic dysfunction during angiography [12]. This exploration aims to unravel the pathophysiological mechanisms and

clinical consequences of this intricate relationship [13]. Risk assessment in patients with Wellens syndrome undergoing angiography extends beyond the procedural context, influencing acute management decisions and holding prognostic implications [14]. Identification of LV systolic dysfunction becomes a pivotal factor in tailoring therapeutic interventions and optimizing post-procedural care for these high-risk individuals [15]. Understanding the evolving landscape of interventions for LV systolic dysfunction in Wellens syndrome patients is essential for improving long-term outcomes [16]. A study by Tiwari et al. conducted on acute coronary syndrome (ACS) patients presenting with Wellens' pattern on electrocardiography reported the 61% frequency of LV systolic dysfunction in patients presented with Wellens syndrome [17]. The rationale of the study is to evaluate the frequency of LV systolic dysfunction in patients with Wellens syndrome undergoing angiography. LV systolic dysfunction is a high-risk manifestation of coronary artery disease and forms the majority of patients suffering from acute myocardial infarction. It is important to recognize the presence of LV systolic dysfunction in patients suffering from Wellens syndrome undergoing angiography to reach early diagnosis and provide prompt treatment to reduce mortality. International and local data is scarce on this topic.

Objective

To evaluate the frequency of left ventricular systolic dysfunction in patients with Wellens syndrome undergoing angiography.

METHODOLOGY

This Descriptive cross-sectional study was conducted at In-patient department of NICVD, Karachi, Pakistan from March 2025 to 10 June 2025. The sample size was calculated using the OpenEpi online software. Based on the findings of Tiwari et al., who reported a 61% frequency of left ventricular systolic dysfunction in patients presenting with Wellens syndrome, and by applying a 95% confidence level with a 7% margin of error, the minimum required sample size was estimated to be 187 [17]. A non-probability consecutive sampling technique was employed.

Inclusion Criteria

- Patients aged 18 to 60 years.
- Both male and female patients.
- All patients undergoing coronary catheterization during the study period.

Exclusion Criteria

- Patients with pre-established left ventricular systolic dysfunction.
- Patients who declined to provide informed consent.

Data Collection

The study was initiated after obtaining approval from the Research Evaluation Unit (REU) of the College of Physicians and Surgeons Pakistan (CPSP) and the institutional review board of NICVD. Written informed consent was obtained from all patients meeting the inclusion criteria. For each eligible patient, demographic data including name, gender, and age were recorded. Anthropometric measurements were taken using a digital weighing machine and stadiometer, and body mass index (BMI) was calculated (BMI = weight/height^2). Relevant medical history was documented, including diabetes mellitus, hypertension, smoking status, and family history of ischemic heart disease (IHD) and dilated cardiomyopathy (DCMP). Investigations such as electrocardiogram (ECG), echocardiography (ECHO), and coronary angiography were performed by postgraduate trainees in cardiology or consultant cardiologists trained according to standard protocols. Left ventricular ejection fraction (LVEF) was assessed through echocardiography. Data were recorded in a pre-defined proforma.

Data Analysis

All data were analyzed using IBM SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables such as age, weight, height, BMI, duration of diabetes mellitus, duration of hypertension, duration of smoking, Troponin I levels, and LVEF (%) were expressed as mean ± standard deviation (SD). If normal distribution was not assumed, the data were presented as median with interquartile range (IQR). Categorical variables including gender, age groups, diabetes mellitus, hypertension, smoking, family history of IHD/DCMP, number of vessels involved, and the presence of LV systolic dysfunction were expressed as frequencies and percentages. Effect modifiers such as gender, diabetes, hypertension, smoking, family history of IHD/DCMP, and number of involved vessels were controlled through stratification. The chi-square test was applied, and a p-value ≤ 0.05 was considered statistically significant.

RESULTS

A total of 187 patients with Wellens syndrome were included. The mean age of the cohort was 51.3 ± 7.8 years, with most patients (52.4%) in the 51–60-year age group, followed by 34.8% in the 41–50-year range, and 12.8% between 18–40 years. Males constituted a larger proportion (58.8%) compared to females (41.2%). The mean BMI was 26.8 ± 3.9 kg/m^2, indicating an overall overweight population. Nearly half of the patients were diabetic (49.2%), while 54.0% had hypertension and 41.2% had a history of smoking. A family history of ischemic heart disease was reported in 23.0% of patients, whereas a family history of dilated cardiomyopathy was less common (6.4%). Electrocardiographic analysis revealed biphasic T-wave changes in 64.2% of patients, while deeply inverted T waves were observed in 35.8%. On coronary angiography, single-vessel disease was noted in 33.2%, double-vessel disease in 38.0%, and triple-vessel disease in 28.9%. The culprit lesion most frequently involved the proximal LAD, found in 87.7% of cases.

Table 1

Baseline Demographic and Clinical Characteristics (N = 187)

Variable	Value
Age, years (mean ± SD)	51.3 ± 7.8
18–40 years	24 (12.8%)
41–50 years	65 (34.8%)
51–60 years	98 (52.4%)
Gender (Male)	110 (58.8%)
Gender (Female)	77 (41.2%)

BMI, kg/m^2 (mean ± SD)	26.8 ± 3.9
Diabetes Mellitus	92 (49.2%)
Hypertension	101 (54.0%)
Smoking	77 (41.2%)
Family History of IHD	43 (23.0%)
Family History of DCMP	12 (6.4%)
ECG findings	
Biphasic T-wave changes	120 (64.2%)
Deeply inverted T waves	67 (35.8%)
Single-vessel disease	62 (33.2%)
Double-vessel disease	71 (38.0%)
Triple-vessel disease	54 (28.9%)
Proximal LAD lesion	164 (87.7%)

Assessment of left ventricular systolic function showed that 59.4% of patients had LVSD, while 40.6% maintained preserved systolic function. Among those with LV dysfunction, 25.7% had mild impairment (LVEF 40–49%), 20.9% had moderate dysfunction (LVEF 30–39%), and 12.8% had severe dysfunction (LVEF <30%).

Table 2

Frequency and Severity of Left Ventricular Systolic Dysfunction

LV Systolic Dysfunction	Frequency n (%)
Present	111 (59.4%)
Absent	76 (40.6%)
Mild (LVEF 40–49%)	48 (25.7%)
Moderate (LVEF 30–39%)	39 (20.9%)
Severe (LVEF <30%)	24 (12.8%)

Stratified analysis demonstrated that LVSD was slightly more common in males than females (62.7% vs. 55.6%), although this difference was not statistically significant (p = 0.28). In contrast, diabetes mellitus was significantly associated with LVSD, with 68.5% of diabetics affected compared to 50.0% of non-diabetics (p = 0.01). Similarly, hypertensive patients showed a higher frequency of LVSD (65.3% vs. 52.3%, p = 0.04). The number of diseased vessels also correlated strongly with systolic dysfunction. LVSD was present in 45.2% of patients with single-vessel disease, 57.7% of those with double-vessel disease, and 77.8% of patients with triple-vessel disease, with the association being highly significant (p < 0.001).

Table 3

Stratified Analysis by Gender

Gender	LVSD Present n (%)	p-value
Male (n=110)	69 (62.7%)	0.28
Female (n=77)	43 (55.6%)	
LVSD Present n (%)		
Diabetes Mellitus Present (n=92)	63 (68.5%)	0.01*
Diabetes Mellitus Absent (n=95)	48 (50.0%)	
Hypertension Present (n=101)	66 (65.3%)	0.04*
Hypertension Absent (n=86)	45 (52.3%)	
Number of Vessels		
Single-vessel disease (n=62)	28 (45.2%)	<0.001*
Double-vessel disease (n=71)	41 (57.7%)	
Triple-vessel disease (n=54)	42 (77.8%)	

DISCUSSION

This study aimed to determine the frequency of left ventricular systolic dysfunction (LVSD) among patients with Wellens syndrome undergoing coronary angiography at a tertiary care cardiac center. The findings revealed that 59.4% of patients had LVSD, with varying degrees of severity ranging from mild to severe. This indicates that a substantial proportion of patients presenting with Wellens syndrome already demonstrate impaired ventricular contractility at the time of angiographic evaluation. The observed frequency of LVSD in our cohort aligns with previous research, which has consistently reported a high burden of ventricular dysfunction among patients with critical proximal LAD disease. Prior studies have suggested that nearly two-thirds of patients with Wellens syndrome develop LV systolic impairment, reflecting the extent of ischemic injury associated with delayed or incomplete revascularization. The predominance of proximal LAD involvement in our study (87.7%) further supports this association, as LAD occlusion is known to compromise a large portion of the left ventricle, leading to a higher risk of dysfunction [18].

Our study also highlighted important clinical associations. Patients with diabetes mellitus demonstrated a significantly higher frequency of LVSD compared to non-diabetics (68.5% vs. 50.0%, p = 0.01). This finding is consistent with earlier evidence that diabetes accelerates atherosclerosis, worsens microvascular dysfunction, and predisposes to adverse ventricular remodeling. Similarly, hypertensive patients had higher rates of LVSD (65.3% vs. 52.3%, p = 0.04), underscoring the role of chronic pressure overload in compromising left ventricular function when combined with ischemic injury [19]. These associations emphasize the compounded risk carried by traditional cardiovascular comorbidities in Wellens patients. Interestingly, smoking status did not show a statistically significant relationship with LVSD in our population (p = 0.72). While smoking is a well-established risk factor for coronary artery disease, its direct contribution to acute LV dysfunction in Wellens syndrome appears less pronounced when compared to factors like diabetes and hypertension. Nevertheless, the high prevalence of smoking (41.2%) in this cohort remains concerning and likely contributes to overall disease burden [20].

Another key finding was the significant association between the number of vessels involved and the presence of LVSD. Triple-vessel disease patients exhibited the highest prevalence of LV dysfunction (77.8%), compared to single-vessel disease patients (45.2%). This demonstrates that disease severity and extent of coronary involvement are strongly linked with ventricular performance [21]. Previous research has similarly shown that multivessel coronary artery disease predicts worse outcomes and more severe systolic dysfunction in ischemic cardiomyopathy. From a clinical standpoint, the high burden of LVSD among Wellens patients underscores the importance of early recognition and timely intervention [22]. Wellens syndrome is often identified in a clinically stable state, with minimal biomarker elevation and without significant ST-segment elevation. However, as our results highlight, a large proportion of these patients already have LV systolic impairment at presentation. This finding reinforces the principle that conservative management or delayed revascularization should be avoided, and urgent angiography with definitive intervention should be prioritized [23]. A strength of this study is its focus on a high-risk yet underreported subset

of acute coronary syndrome patients in a local population, providing valuable regional data. Moreover, echocardiographic assessment of LVEF was performed according to standard protocols, ensuring reliability of ventricular function evaluation. However, certain limitations should be acknowledged. The cross-sectional design precludes assessment of long-term outcomes such as progression to heart failure or mortality. Additionally, the use of a single-center, non-probability sampling approach may limit generalizability. Finally, unmeasured confounders such as medication adherence, glycemic control, and duration of hypertension may have influenced the observed associations.

CONCLUSION

It is concluded that left ventricular systolic dysfunction is highly prevalent in patients with Wellens syndrome undergoing coronary angiography, with nearly six out of ten patients demonstrating impaired ventricular function. The frequency of LVSD was significantly higher among patients with diabetes mellitus, hypertension, and multivessel coronary artery disease, highlighting the compounded risk carried by these comorbidities. These findings underscore the prognostic importance of early recognition and echocardiographic assessment of ventricular function in Wellens syndrome. Prompt angiographic evaluation and timely revascularization remain crucial to prevent further deterioration of left ventricular performance and to improve clinical outcomes in this high-risk population.

REFERENCES

1. Habib, M. (2024). Right ventricle function among patients with Wellens syndrome versus de winter syndrome. *Journal of Cardiovascular and Cardiology*, 1-3. https://doi.org/10.61440/jcc.2024.v2.22
2. Zhou, L., Gong, X., Dong, T., Cui, H., Chen, H., & Li, H. (2022). Wellens' syndrome: Incidence, characteristics, and long-term clinical outcomes. *BMC Cardiovascular Disorders*, *22*(1). https://doi.org/10.1186/s12872-022-02560-6
3. Han, D., Tamarappoo, B., Klein, E., Tyler, J., Chakravarty, T., Otaki, Y., ... & Friedman, J. (2021). Computed tomography angiography-derived extracellular volume fraction predicts early recovery of left ventricular systolic function after transcatheter aortic valve replacement. *European Heart Journal-Cardiovascular Imaging*, *22*(2), 179-185. https://doi.org/10.1093/ehjci/jeaa310
4. Van de Werf, F., Bax, J., Betriu, A., Blomstrom-Lundqvist, C., Crea, F., Falk, V., Filippatos, G., Fox, K., Huber, K., Kastrati, A., Rosengren, A., Steg, P. G., Tubaro, M., Verheugt, F., Weidinger, F., Weis, M., & Evseev, M. O. (2009). Management of acute myocardial infarction in patients presenting with persistent St-segment elevation. *Rational Pharmacotherapy in Cardiology*, *5*(5), 73-89. https://doi.org/10.20996/1819-6446-2009-5-5-73-89
5. De Zwaan, C., Bär, F. W., Janssen, J. H., Cheriex, E. C., Dassen, W. R., Brugada, P., Penn, O. C., & Wellens, H. J. (1989). Angiographic and clinical characteristics of patients with unstable angina showing an ECG pattern indicating critical narrowing of the proximal LAD coronary artery. *American Heart Journal*, *117*(3), 657-665. https://doi.org/10.1016/0002-8703(89)90742-4
6. Zhou, D., Yan, M., Cheng, Q., Feng, X., Tang, S., & Feng, Y. (2022). Prevalence and prognosis of left ventricular diastolic dysfunction in community hypertension patients. *BMC Cardiovascular Disorders*, *22*(1). https://doi.org/10.1186/s12872-022-02709-3
7. Marchi, E., Muraca, I., Cesarini, D., Pennesi, M., & Valenti, R. (2024). Double coronary artery occlusion presenting as inferior ST segment elevation myocardial infarction and Wellens syndrome type a: A case report. *European Heart Journal - Case Reports*, *8*(8). https://doi.org/10.1093/ehjcr/ytae394
8. Chartrand-Lefebvre, C., Cadrin-Chênevert, A., Bordeleau, E., Ugolini, P., Ouellet, R., Sablayrolles, J., & Prenovault, J. (2007). Coronary computed tomography angiography: overview of technical aspects, current concepts, and perspectives. *Journal-Canadian Association of Radiologists*, *58*(2), 92.
9. Patel, K., Alattar, F., Koneru, J., & Shamoon, F. (2014). ST-elevation myocardial infarction after pharmacologic Persantine stress test in a patient with Wellens' syndrome. *Case Reports in Emergency Medicine*, *2014*, 1-3. https://doi.org/10.1155/2014/530451
10. Miner B, Grigg WS, Hart EH. Wellens Syndrome. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan.
11. Crea, F., & Libby, P. (2017). Acute coronary syndromes. *Circulation*, *136*(12), 1155-1166. https://doi.org/10.1161/circulationaha.117.029870
12. Garg, A., Rout, A., Raheja, H., Hakeem, H., & Sharma, S. (2020). Long-term follow-up of percutaneous coronary intervention versus coronary artery bypass grafting in left main coronary artery disease: A systematic review and meta-analysis. *Catheterization and Cardiovascular Interventions*, *98*(3), 427-433. https://doi.org/10.1002/ccd.29338
13. Antman, E., Bassand, J., Klein, W., Ohman, M., Lopez Sendon, J. L., Rydén, L., Simoons, M., & Tendera, M. (2000). Myocardial infarction redefined—a consensus document of the joint European Society of Cardiology/American College of Cardiology committee for the redefinition of myocardial infarction. *Journal of the American College of Cardiology*, *36*(3), 959-969. https://doi.org/10.1016/s0735-1097(00)00804-4
14. Ji, L., Su, X., Qin, W., Mi, X., Liu, F., Tang, X., Li, Z., & Yang, L. (2015). Novel risk score of contrast-induced nephropathy after percutaneous coronary intervention. *Nephrology*, *20*(8), 544-551. https://doi.org/10.1111/nep.12429
15. König, S., Hohenstein, S., Nitsche, A., Pellissier, V., Leiner, J., Stellmacher, L., Hindricks, G., & Bollmann, A. (2023). Artificial intelligence-based identification of left ventricular systolic dysfunction from 12-lead electrocardiograms: External validation and advanced application of an existing model. *European Heart Journal - Digital Health*, *5*(2), 144-151. https://doi.org/10.1093/ehjdh/ztad081
16. Marchi, E., Muraca, I., Cesarini, D., Pennesi, M., & Valenti, R. (2024). Double coronary artery occlusion presenting as inferior ST segment elevation myocardial infarction and Wellens syndrome type a: A case report. *European Heart Journal - Case Reports*, *8*(8). https://doi.org/10.1093/ehjcr/ytae394
17. Tiwari, D. K., & Upadhyaya Belle, C. (2024). 70 Retrospective analysis of acute coronary syndrome (ACS) presenting with wellen's pattern on electrocardiography (ECG). *Acute*

coronary syndromes & interventional cardiology, A75.2-A76. https://doi.org/10.1136/heartjnl-2024-bcs.70

18. Stankovic, I., Kafedzic, S., Janicijevic, A., Cvjetan, R., Vulovic, T., Jankovic, M., Ilic, I., Putnikovic, B., & Neskovic, A. N. (2017). T-wave changes in patients with Wellens syndrome are associated with increased myocardial mechanical and electrical dispersion. *The International Journal of Cardiovascular Imaging*, *33*(10), 1541-1549. https://doi.org/10.1007/s10554-017-1181-4
19. Bin Abdul Malik, M. H., Khan, N. N., Chowdhari, M. J., Rafiq, M. K., Ali, M., LNU, S., Ramzan, H. S., Ahmed, M. M., & Khatoon, F. (2025). Evaluating the impact of Tirzepatide on clinical outcomes in patients with heart failure. *Cureus*. https://doi.org/10.7759/cureus.82118
20. Anwar, S., Rehman, A. U., Hussain, A., Abid, A. R., Saeed, S. ,., & Ramzan, H. S. (2022). Assessment of serum levels of oxidative stress markers and heat shock proteins in myocardial infraction patients. *Pakistan Journal of Medical and Health Sciences*, *16*(6), 912-915. https://doi.org/10.53350/pjmhs22166912
21. Lorca, R., Pascual, I., Aparicio, A., Junco-Vicente, A., Alvarez-Velasco, R., Barja, N., Roces, L., Suárez-Cuervo, A., Diaz, R., Moris, C., Hernandez-Vaquero, D., & Avanzas, P. (2021). Premature STEMI in men and women: Current clinical features and improvements in management and prognosis. *Journal of Clinical Medicine*, *10*(6), 1314. https://doi.org/10.3390/jcm10061314
22. Petropoulos, T., Rooprai, J., Kotowycz, M. A., & Madan, M. (2022). Spontaneous coronary artery dissection in a 50-year-old woman. *Canadian Medical Association Journal*, *194*(15), E549-E553. https://doi.org/10.1503/cmaj.212009
23. Gad, M. M., Mahmoud, A. N., Saad, A. M., Bazarbashi, N., Ahuja, K. R., Karrthik, A. K., Elgendy, I. Y., Elgendy, A. Y., Don, C. W., Hira, R. S., Massoomi, M. R., Saw, J., Anderson, R. D., & Kapadia, S. R. (2020). Incidence, clinical presentation, and causes of 30-Day readmission following hospitalization with spontaneous coronary artery dissection. *JACC: Cardiovascular Interventions*, *13*(8), 921-932. https://doi.org/10.1016/j.jcin.2019.12.033