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ORIGINAL RESEARCH

A Comprehensive Analysis of Clinical, Demographic, Risk Factor, and Echocardiography Profiles of Coronary Slow Flow Phenomenon from Somalia's Largest PCI Center

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Introduction: The coronary slow flow phenomenon (CSFP) is delayed distal vessel opacification in the absence of significant epicardial coronary disease.

Methods: A retrospective analysis was conducted on 103 patients' medical records from June 2022 to January 2024 at the Mogadishu Somali Turkish Training and Research Hospital in Somalia. The study focused on patient demographics, risk factors, echocardiography profiles, and variables related to coronary slow flow such as laboratory tests.

Results: Slow coronary flow was observed in different combinations of coronary arteries, with isolated slow flow in 17 patients and involvement of all three major arteries in 9 patients. Hypertension was significantly higher in the slow coronary flow group compared to the normal coronary artery group. LDL cholesterol levels and left ventricular ejection fraction were statistically significantly lower in patients with slow coronary flow compared to those with normal coronary arteries. The eosinophil count was found to be statistically significantly lower in the slow coronary flow group compared to the normal coronary artery group.

Conclusion: The study on coronary slow flow conducted in Somalia highlights several significant findings. Overall, this study sheds light on the clinical characteristics and potential risk factors associated with coronary slow flow in the Somali population.

Keywords: coronary slow follow, angiography, echocardiography

Introduction

The coronary slow flow phenomenon (CSFP) is defined as delayed distal vessel opacification in the absence of significant epicardial coronary disease.[1] It is an angiographic diagnosis defined by a slow flow of contrast agents in the normal or near-normal epicardial coronary arteries.[2] Approximately 1% of individuals who undergo coronary angiography (CAG) for stable angina pectoris are predicted to have slow coronary flow.[3] Although the pathophysiologic mechanisms involved in CSFP are not yet fully understood, they have been linked to underlying endothelial dysfunction, early atherosclerosis, microvascular disease, platelet dysfunction, alterations in hemorheology, oxidative stress, and both local and systemic inflammatory responses.[4–6]

It is important to note that the CSFP may increase the risk of severe cardiovascular adverse events, and these patients may present with different types of coronary artery diseases, like stable CAD or ACS.[7,8]

A study described the clinical and demographic findings and the presence of common atherosclerosis risk factors in patients with SCF, providing valuable insights into the characteristics of individuals affected by this condition.[9,10] Understanding the clinical, demographic, and risk factor profiles of those affected by this phenomenon is critical for developing effective management strategies and providing better patient care. It has been implicated in cardiovascular risk factors like hypertension, hematological factors such as full blood count parameters, and echocardiographic findings like

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diastolic dysfunction. We studied coronary slowness, which is more common in those who underwent angiography, in Somalia's largest percutaneous coronary intervention (PCI) center. To our knowledge, this is the first study on this topic in the Somali population, focusing on factors like full blood count parameters and echocardiography.

Methodology

Study Design and Data Collection

The study was a retrospective observational study conducted in the cardiology department of the Mogadishu-Somalia Turkish Training and Research Hospital. The study included individuals who had no abnormalities in their angiogram and exhibited coronary slow flow. Individuals with coronary lesions identified during angiography and those with incomplete data were excluded from the study. The study focused on assessing risk factors and echocardiographic findings in patients with slow follow-in angiography. Data extraction was conducted using the FONET system, an electronic health record (EHR) management system used by our hospital. The FONET system is designed to handle comprehensive patient information, including demographics, medical history, treatment plans, and clinical notes. Patients with angiography from June 2022 to January 2024 were enrolled.

Coronary angiography involves imaging the coronary anatomy under fluoroscopy, which is made possible by the direct injection of contrast material into the pericardial coronary arteries through a catheter advanced from a peripheral artery to the aortic root and into the coronary Ostia.

Visual or quantitative coronary angiography was used as it is a simple, easy, and quick way to measure how bad a lesion is.

Hyperlipidemia was a history of dyslipidemia that had been medically diagnosed and/or treated, total cholesterol above 200 mg/dl, low-density lipoprotein above or equal to 130 mg/dl, or high-density lipoprotein below 40 mg/dl. The hypertension criteria were those with a history of previous hypertension, treated with lifestyle changes, and those with a blood pressure greater than 140 mmHg systolic or 90 mmHg diastolic on at least two occasions. Diabetes was defined as a fasting blood sugar level of 126 mg/dl or higher or a history of diabetes diagnosed and/or treated with medication and/or lifestyle change. In our study, smokers were defined as current smokers or those who had recently quit smoking.

According to the American Society of Echocardiography guidelines, left ventricular (LV) measurements were obtained at end-diastole and end-systole, with each parameter's value averaged across three cardiac cycles. The parameters evaluated included ventricular septal thickness at end-diastole (VSTd), LV internal diameter at end-diastole (LVTDd) and end-systole, and posterior wall thickness at end-diastole (PWTd). The assessment of left ventricular diastolic dysfunction (LVDD) utilized transmitral Doppler flow velocities, mitral annular e′ velocity, E/e′ ratio, peak velocity of the tricuspid regurgitation (TR) jet, and left atrial (LA) maximum volume index.[11]

The left ventricular ejection fraction (LVEF) was determined using the modified biplane Simpson's method from apical four-chamber and two-chamber views. To obtain the maximum filling velocities, the pulsed Doppler sampling volume was positioned between the tips of the mitral valve leaflets. Measurements were taken for early diastolic flow (E), the atrial contraction signal (A), and the E-wave deceleration.[12].Echocardiogram results were derived from final reports written by a cardiologist with specialized training in echocardiography.

The BS 2000 Mandary machine was used for hematology parameters.

Ethical Consideration

Since our hospital is a research hospital, generally informed consent is obtained from every patient admitted to obtaining their data for retrospective research purposes from the hospital medical records, and this study did not disclose any personal information. The study was approved by the research ethics committee of Mogadishu Somali Turkey Training and Research Hospital (Ethics Protocol No: MSTH/16340). The study was performed in line with the principles of the Declaration of Helsinki.

Statistical Analysis

Data were analyzed using a commercially available statistics software package (SPSS® for Windows). The Kolmogorov–Smirnov test was used to assess whether the distribution of variables was normal. Descriptive statistical methods (mean, standard deviation, frequency, ratio, minimum, maximum) were used when evaluating the study data. The One-way ANOVA test and Kruskal–Wallis test were used to compare normally distributed and non-normal distributed continuous variables, respectively. The Pearson chi-square was used for the evaluation of categorical data. $P < 0.05$ was considered as statistically meaningful.

Result

Between June 2022 to January 2024, a total of 480 angiographies performed at Mogadishu Turkish-Somali Education and Research Hospital were evaluated. Among these 41 were identified with slow coronary flow and compared to 42 as a control group. In the group with the slow coronary flow, isolated slow flow was observed in 17 patients (RCA n: 3, LAD n: 14), with no isolated CX involvement observed. Slow flow in all three major coronary arteries was seen in 9 patients, while in 15 patients, slow flow was observed in two arteries (LAD-RCA n: 5, LAD-CX n: 10). The percentage distributions of patients in the slow coronary flow group are shown in Figure 1.

No differences were found in terms of age and gender distribution among the patients included in the study, with p-values of 0.675 and 0.934, respectively. Regarding coronary artery risk factors, hypertension was significantly higher in the slow coronary flow group compared to the normal coronary artery group (19.5% vs 4.8%; p: 0.043). The demographic characteristics of the patients included in the study are shown in Table 1.

As shown in Table 2, when the echocardiograms of the patients included in the study were evaluated, no moderate-to-severe valve pathologies were detected in either group. Left ventricular diastolic dysfunction was observed in 11 patients

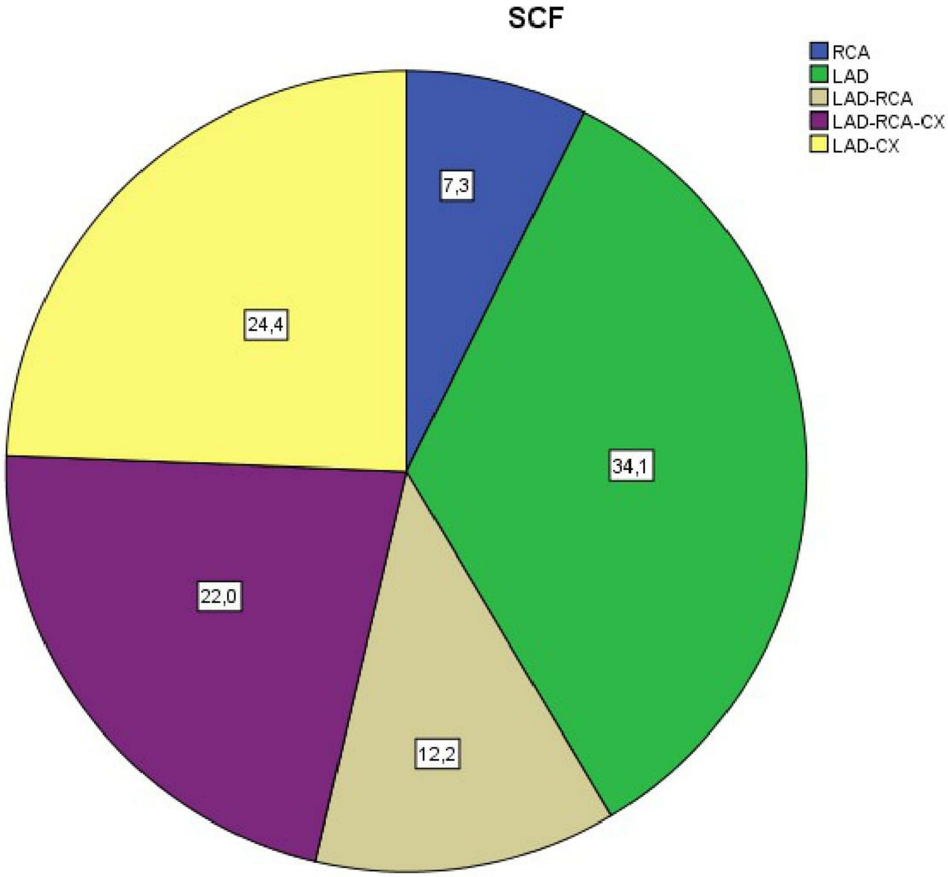

Figure 1 Shows % of CSF in terms of number of involved vessels.

Table 1 Demographic Characteristics Among Case and Control Group

	Slow Flow Group (n=41)	Normal Coronary Artery Group (n=42)	p
Age [yr]	57 ± 15	58 ± 13	0.675
Gender: F / M (n)	15 / 26	15 / 27	0.934
Smoking, n (%)	26 (63)	23 (54)	0.482
Hypertension, n (%)	8 (19.5)	2 (4.8)	0.043*
Type 2 diabetes, n (%)	13 (31.7)	20 (47.6)	0.091
Dyslipidemia n (%)	9 (22)	12 (28.6)	0.406

Notes: Data are mean ± standard deviation (SD) or the number of patients and percentage. *Significant association detected.

Table 2 Laboratory Parameters of Patients with Coronary Slow Flow and Controls

	Slow Flow Group (n=41)	Normal Coronary Artery Group (n=42)	P
EF %	40 (20–65)	52 (20–66)	0.033*
DD n (%)	11 (26.8)	5 (11.9)	0.085
LDL-C (mg/dL)	99.76 ± 45.83	121.57 ± 37.15	0.019*
HDL-C (mg/dL)	44.5 (21–76)	45 (24–70)	0.763
Triglyceride (mg/dL)	119.5 (54–313)	135 (28–356)	0.763
ALT (U/L)	23.5 (6–44)	18 (6–45)	0.241
AST (U/L)	24.5 (14–74)	22 (12–77)	0.273
K (mmol/L)	4 (2.8–5.7)	4 (3.4–6.5)	0.245
Na (mmol/L)	140 (125 −147)	139.5 (119–146)	0.227
Urea	24 (8–112)	27 (8–137)	0.312
Creatina (mg/dL)	0.6 (0.1–2.6)	0.8 (0.4–11)	0.617
BASO (x1000/mm^3)	0.3 (0.04–1.1)	0.2 (0.1–1)	0.754
ESO (%)	1.65 (0.1–13)	2.9 (1.1–14.4)	0.001*
HGB (g/dL)	13.36 ± 1.91	12.6 ± 2.18	0.095
NEUT (x1000/mm^3)	58.77 ± 16.59	56.62 ± 10.98	0.377
PLT (x1000/mm^3)	265 ± 102	300 ± 92	0.102
HbAc1 (%)	5.6 (4.4–13.6)	5.95 (3.4–12.7)	0.996

Notes: Data are mean ± standard deviation (SD) or median (minimum – maximum). * Significant association detected.

Abbreviations: EF, ejection fraction; DD, diastolic dysfunction; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ALT, alanine aminotransferase; AST, aspartate aminotransferase; K, Potassium; Na, sodium; BASO, basophil; ESO, eosinophil; HGB, hemoglobin; NEUT, neutrophil; PLT, platelet; HbAc1, glycated haemoglobin.

in the slow coronary flow group and 5 patients in the normal coronary artery group, with no statistically significant difference between the groups. However, the left ventricular ejection fraction was found to be statistically significantly lower in the slow coronary flow group compared to the normal coronary artery group.

While no difference was observed in terms of dyslipidemia between the two groups, LDL cholesterol levels were found to be statistically significantly lower in patients with slow coronary flow compared to those with normal coronary arteries. Other biochemical parameters were found to be at similar levels in both groups.

Hemoglobin levels, basophil, neutrophil, and platelet counts were similar in both groups. However, eosinophil count was statistically significantly lower in the slow coronary flow group compared to the normal coronary artery group.

Discussion

Studies on CSF were conducted to identify clinical characteristics, risk factors, and prognosis related to the condition. It is more prevalent among male patients.[13]

Extensive research has been conducted on the risk factors linked to the coronary slow flow phenomenon (CSFP). CSFP has been linked to independent risk variables such as smoking, dyslipidemia, and hypertension and occurs in 1–5% of diagnostic coronary angiography patients, particularly young male smokers.[14,15] Similar to other previously conducted studies, we found a correlation between coronary slow flow and high blood pressure.

This study also discovered a weak connection between low levels of LDL cholesterol and the occurrence of coronary slow flow, which contradicts previous investigations' findings.[16]

Furthermore, The CSFP is a significant angiographic finding that has been linked to microvascular dysfunction and has implications for patient outcomes.[17] In other research, the coronary slow flow (CSF) phenomenon has been linked to both systolic and diastolic abnormalities in left ventricular (LV) function.[18] Similar to our findings, these studies demonstrate a correlation between reduced systolic ejection fraction and the presence of coronary slow flow.

The relationship between left ventricular ejection fraction and coronary slow flow was investigated in patients with coronary microvascular disease and obstructive coronary artery disease.[19]

The relationship between coronary slow flow and low ejection fraction is complicated and multifaceted, involving coronary microvascular dysfunction, coronary artery disease, and their impact on left ventricular function.[20]

Although some studies have not identified a statistically significant relationship between traditional cardiovascular risk factors and the coronary slow flow phenomenon, others have pointed out the association of atherosclerosis, vascular spasm, and microvascular dysfunction with the CHA2DS2-VASc score, which is commonly used to assess risk for stroke.[21,22]

Complete blood cell count components have been demonstrated to be a valid and easily available predictor of cardiovascular events, especially among patients with coronary artery disease.[23]

Regarding this, the CSFP group exhibited higher eosinophil counts compared to the control group, consistent with findings from other studies.[24] Eosinophils contribute to the activation of the coagulation system and platelets, as well as promoting inflammation, endothelial dysfunction, and aneurysm formation. Additionally, they play a significant role in vascular injury.[25]

Additionally, the neutrophil/lymphocyte ratio has been presented as a potential predictor of coronary artery disease, coronary artery ectasia, and coronary slow flow, suggesting a probable relationship between khat consumption and coronary slow flow.[26]

Although our study did not encompass this particular risk factor, it's essential to acknowledge the elevated prevalence of khat consumption within this community, especially among those with coronary artery disease, warranting further study in those with coronary slow follow-up too.

Limitation

Although this was the first study of coronary slow-following in Somalia, the study's limitations were clear. The following are some limitations of our research: First, it is a retrospective study conducted at a single center. Second, the number of participants is small, so the results cannot be generalized. Finally, the lack of comparable results for Coronary Slow Follow in other African nations limits the study's wider context.

Conclusion

Our comprehensive analysis offers illuminates the complex interplay of clinical, demographic, risk factor, and echocardiography profiles among patients experiencing the Coronary Slow Flow Phenomenon (CSFP) at Somalia's largest PCI center. We discovered key patterns and associations while meticulously examining a diverse patient population, providing valuable insights into the understanding and management of this enigmatic condition. Given the multifactorial nature of CSFP's presentation, our findings highlight the importance of tailored approaches to diagnosis and treatment. Furthermore, our findings emphasize the need for additional research to better understand the underlying mechanisms driving CSFP and to investigate potential novel therapeutic interventions. This study contributes to global efforts to improve patient care and outcomes in the field of coronary artery disease by increasing our understanding of CSFP in the Somali community.

Author Contributions

All authors made a significant contribution to this work, whether that is, in the conception, study design, execution, acquisition of data, analysis, and interpretation, or all these areas; took part in drafting, revising, or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors declare that they have no conflicts of interest.

References

1. Beltrame JF, Limaye SB, Horowitz JD. The coronary slow flow phenomenon–a new coronary microvascular disorder. *Cardiology*. 2002;97(4):197–202. doi:10.1159/000063121
2. Wang Y, Ma C, Zhang Y, et al. Assessment of left and right ventricular diastolic and systolic functions using two-dimensional speckle-tracking echocardiography in patients with coronary slow-flow phenomenon. *PLoS One*. 2015;10(2):e0117979. doi:10.1371/journal.pone.0117979
3. Goel PK, Gupta SK, Agarwal A, et al. Slow coronary flow: a distinct angiographic subgroup in syndrome X. *Angiology*. 2001;52(8):507–514. doi:10.1177/000331970105200801
4. Ghaffari S, Tajlil A, Aslanabadi N, et al. Clinical and laboratory predictors of coronary slow flow in coronary angiography. *Perfusion*. 2017;32(1):13–19. doi:10.1177/0267659116659918
5. Alvarez C, Henry SIU. Coronary slow-flow phenomenon as an underrecognized and treatable source of chest pain: case series and literature review. *J Investig Med High Imp Case Rep*. 2018;6:2324709618789194. doi:10.1177/2324709618789194
6. Shui Z, Wang Y, Sun M, et al. The effect of coronary slow flow on left atrial structure and function. *Sci Rep*. 2021;11(1):7511. doi:10.1038/s41598-021-87193-z
7. Canpolat U, Çetin EH, Cetin S, et al. Association of monocyte-to-HDL cholesterol ratio with slow coronary flow is linked to systemic inflammation. *Clin App Thrombosis*. 2016;22(5):476–482. doi:10.1177/1076029615594002
8. Aparicio A, Cuevas J, Morís C, et al. Slow coronary blood flow: pathogenesis and clinical implications. *Europ Cardiol Rev*. 2022;17:1.
9. Abrar U, Shaikh JK, Ahmed I, et al. Comparison of the outcomes between coronary no-reflow and slow-flow phenomenon in non-stemi patients. *Pak J Med Health Sci*. 2022;16(5):1407. doi:10.53350/pjmhs221651407
10. El Zayat A, Abdelaziz M, Yousry A, et al. Evolution of diastolic dysfunction in patients with coronary slow flow phenomenon and acute non-ST segment elevation myocardial infarction. *J Cardiovas Imag*. 2021;29(4):347. doi:10.4250/jcvi.2020.0185
11. Sanati H, Kiani R, Shakerian F, et al. Coronary slow flow phenomenon clinical findings and predictors. *Res Cardiovasc Med*. 2016;5:1–5.
12. Hassan MS, Mire Waberi M, Osman Sidow N, et al. Analysis of echocardiographic findings of patients with acute ischemic stroke admitted to a tertiary care hospital in Mogadishu. *Internat J Gen Med*. 2023;Volume 16:2887–2895. doi:10.2147/IJGM.S414014
13. Seyyed-Mohammadzad MH, Rashtchizadeh S, Khademvatani K, et al. Ventricular dysfunction in patients with coronary slow-flow phenomenon: a single-center case–control study. *Heart Views*. 2020;21(2):60. doi:10.4103/HEARTVIEWS.HEARTVIEWS_119_18
14. Arjmand N, Dehghani MR. Complete blood cell count components and coronary slow-flow phenomenon. *Therap Clin Risk Manag*. 2016;Volume 12:1827–1829. doi:10.2147/TCRM.S121433
15. Sanghvi S, Mathur R, Baroopal A, et al. Clinical, demographic, risk factor and angiographic profile of coronary slow flow phenomenon: a single centre experience. *Indian Heart J*. 2018;70:S290–S294.
16. Dehghani MR, Rezaei Y, Taghipour-Sani L. Superiority of total white blood cell count over other leukocyte differentials for predicting long-term outcomes in patients with non-ST elevation acute coronary syndrome. *Biomarkers*. 2014;19(5):378–384. doi:10.3109/1354750X.2014.915429
17. Dutta U, Sinha A, Demir OM, et al. Coronary slow flow is not diagnostic of microvascular dysfunction in patients with angina and unobstructed coronary arteries. *J Am Heart Assoc*. 2023;12(1):e027664. doi:10.1161/JAHA.122.027664
18. Gunes Y, Tuncer M, Guntekin U, Ceylan Y, Sahin M, Simsek H. Regional functions of the left ventricle in patients with coronary slow flow and the effects of nebivolol. *Therap Advan Cardiovas Dis*. 2009;3(6):441–446. doi:10.1177/1753944709345926
19. Buono DEL, Giuseppe M, Camilli M, et al. Coronary microvascular dysfunction across the spectrum of cardiovascular diseases: JACC state-of-the-art review. *J Am Coll Cardiol*. 2021;78(13):1352–1371. doi:10.1016/j.jacc.2021.07.042
20. Mayala HA, Mafuru M, Mkangala A, et al. Factors influencing left ventricular ejection fraction in patients with coronary microvascular disease and obstructive coronary artery disease. *BMC Res Notes*. 2020;13(1):1–6. doi:10.1186/s13104-020-05008-2

21. Tosu AR, Kalyoncuoğlu M, Biter H, et al. Association of eosinophil-to-lymphocyte ratio with coronary slow-flow phenomenon in patients undergoing coronary angiography. *Archiv Med Sci*. 2022;2022:1–7.
22. Sadr-Ameli MA, Saedi S, Saedi T, et al. Coronary slow flow: benign or ominous? *Anat J Card*. 2015;15(7):531. doi:10.5152/akd.2014.5578
23. Köseoğlu C, Zorlu Ç. Use of CHA2DS2-VASc score in patients with stable angina pectoris with slow flow detected in coronary angiography. *Cukurova Med J*. 2020;45(3):827–834. doi:10.17826/cumj.675596
24. Altas Y, Kurtoglu E, Yaylak B, et al. The relationship between eosinophilia and slow coronary flow. *Therap Clin Risk Manag*;2015. 1187–1191. doi:10.2147/TCRM.S87761
25. Demir M, Coşar S, Melek M. Evaluation of plasma eosinophil count and mean platelet volume in patients with coronary slow flow. *Clinics*. 2014;69:323–326. doi:10.6061/clinics/2014(05)05
26. Yilmaz M, Korkmaz H, Bilen MN, et al. Could neutrophil/lymphocyte ratio be an indicator of coronary artery disease, coronary artery ectasia and coronary slow flow? *J Int Med Res*. 2016;44(6):1443–1453. doi:10.1177/0300060516664637