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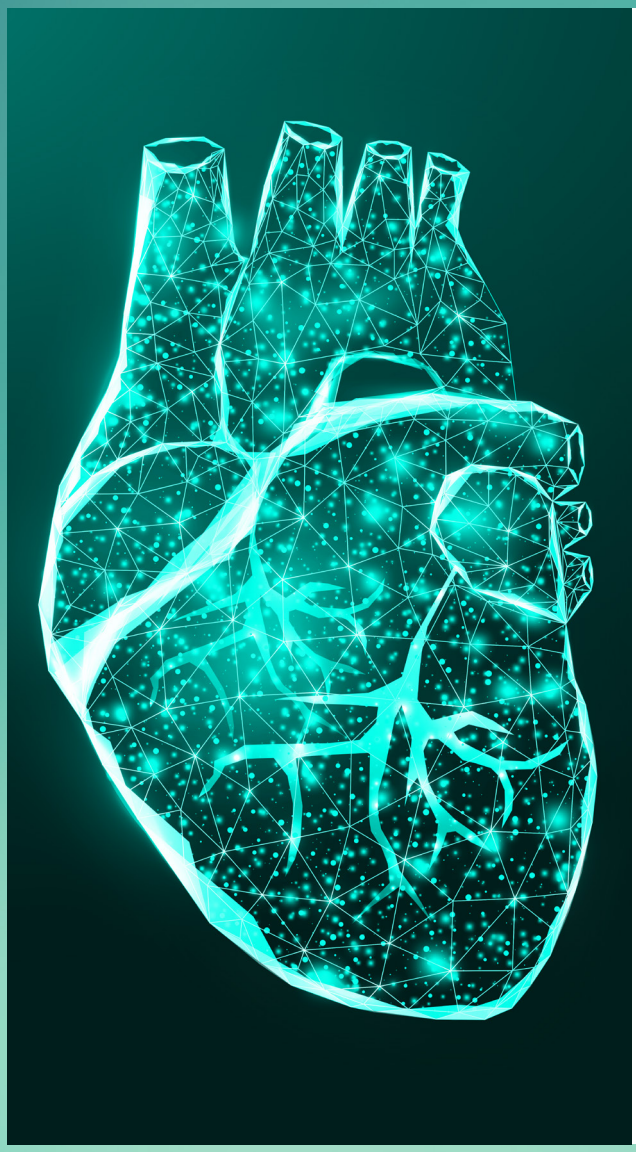

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Authors: Kino Kino, Heka Widya Putri

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International Journal of The Cardiovascular Academy

REVIEW

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Int J Cardiovasc Acad 2025;11(4):147-153

Unveiling the Efficacy and Safety Divide Between Fluoroscopy and Non-fluoroscopy Approaches in Transcatheter Atrial Septal Occluder Procedures: A Systematic Review and Meta-analysis

Kino Kino, Heka Widya Putri

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Abstract

This study compares the effectiveness of fluoroscopy versus non-fluoroscopy procedures during percutaneous closure of atrial septal defects (ASD) in children. The clinical concern surrounding radiation exposure in children and medical staff is well recognized. A systematic review and meta-analysis were conducted using PubMed, ScienceDirect, and Cochrane databases, including studies up to February 2024. Prospective studies were assessed for risk of bias and effect sizes were calculated using standard mean differences (MD) and log risk ratios. Out of 18 studies, five were included in qualitative analysis and four in the meta-analysis. Findings indicated significantly higher success rates in the non-fluoroscopy group compared to the fluoroscopy group [odds ratio (OR) =3.40, $P < 0.001$], shorter procedure times (MD =12.59), and a lower risk of postoperative complications (OR =3.22). Non-fluoroscopy-guided ASD closure appears to be a more effective and safer approach in pediatric patients.

Keywords: Atrial septal defect, procedures, fluoroscopy, pediatric

INTRODUCTION

Atrial septal defect (ASD) is a congenital heart anomaly with the secundum type of ASD comprising the majority of clinically significant cases. While a patent foramen ovale (PFO) may be present in up to 25% of the population, true ASDs have a lower incidence of approximately 1.6 per 1,000 live births. Differentiation between PFO and ASD is crucial in both diagnosis and management.[1,2] Treatment modalities include open cardiac surgery and percutaneous device closure, with the latter being preferred because of its minimally invasive nature and proven efficacy.[3,4]

Fluoroscopy in combination with echocardiography is the traditional method for device guidance during ASD closure. However, this exposes patients and healthcare personnel to ionizing radiation, posing long-term health risks, especially in children.[4,5] This concern is particularly significant for children given their heightened sensitivity to radiation and the potential for long-term side effects over their extensive expected lifespan.[3,6]

With the aim of reducing radiation exposure, some centers have explored using echocardiography alone, either transesophageal echocardiography (TEE) or transthoracic echocardiography (TTE) to guide device closure.[7,8] Despite growing interest,

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non-fluoroscopy-guided closure has not been widely adopted, partly due to the lack of robust comparative evidence. This study aims to systematically compare the safety, effectiveness, and procedural outcomes of fluoroscopy-guided versus non-fluoroscopy-guided transcatheter ASD closure in pediatric patients. We hypothesized that non-fluoroscopy techniques would yield comparable or superior outcomes with reduced complication rates and procedural time.

METHODS

Study Criteria

This systematic review and meta-analysis adhered to Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines and was not registered in PROSPERO. A comprehensive search of PubMed, Cochrane Library, ScienceDirect, Scopus, and Web of Science was conducted through February 2024. We used specific inclusion criteria to identify relevant articles, focusing on primary outcomes such as success rate, mean procedure time, and complication rate. The selection of studies was limited to those published in English. Articles in other languages, duplicates, review articles, and publications not relevant to the topic were not included. No restrictions were placed on the publication year of the selected studies.

Participants Criteria

This study cohort included patients preoperatively diagnosed with isolated type II ASD that required surgery without concomitant heart disease. The patient's medical history, clinical symptoms, chest X-ray, electrocardiogram, and echocardiogram were used to diagnose this condition. Patients were excluded if they exhibited symptoms of an infectious disease, severe pulmonary hypertension, or any other condition that would contraindicate surgery.

Literature Search and Study Selection

We conducted a comprehensive search until February 2024 using PubMed, MEDLINE, Cochrane Library, Scopus, Web of Science, and ScienceDirect, following the PRISMA guidelines. The search terms utilized were "[fluoroscopy odds ratio (OR) fluoroscopic]" and "(atrial septal)" and "(device closure OR transcatheter OR echocardiography OR radiation-free)". After removing duplicates and review articles, the remaining research titles and abstracts were independently examined to determine eligibility. The full texts of selected studies were then evaluated against the inclusion and exclusion criteria.

Statistical Analysis

Effect sizes were calculated using the Mantel-Haenszel method. Because a fixed-effects model was initially employed given the clear clinical heterogeneity among the studies, a random-effects model was ultimately used for pooled estimates. Heterogeneity was assessed using the I^2 statistic and τ^2. Funnel plots were generated to assess publication bias. Data extraction included study characteristics, participant demographics, intervention details, and outcomes. Quality and risk of bias was assessed using the Cochrane Risk of Bias Tool for RCTs and ROBINS-I for non-randomized studies. The quality assessment results of the individual studies are presented in the Table 1.

RESULTS

Out of 98 identified records, eighteen full-text articles were assessed for eligibility, and four studies comprising a total of 1,143 pediatric patients were included in the meta-analysis. The studies were conducted in diverse settings, including China, Switzerland, and Germany. The methodological characteristics and comparative outcomes of these studies are summarized in Table 2. Non-fluoroscopy techniques showed significantly higher procedural success [relative risk (RR): 3.40] [95% confidence interval (CI): 1.92-6.01], $P < 0.001$), shorter procedure durations [mean difference (MD) =-12.59 minutes (95% CI: -16.8 to -8.3)], and higher intraoperative and postoperative complications [OR =3.22 (95% CI: 1.85-5.62)]. Heterogeneity was moderate (I^2 =56%). The method used to choose the studies for this review is visually represented in Figure 1 following the standard PRISMA flow diagram.

DISCUSSION

Four studies involving 1,143 children who underwent transcatheter ASD closure were conducted in China, Switzerland, and Germany. The studies assessed various parameters, including the success rate, procedure duration, and intraoperative and postoperative complications. These findings indicate that non-fluoroscopy methods are more effective than fluoroscopy, with a higher success rate and shorter procedure durations.[9] However, only four studies were included, and one reported an extremely wide CI [Kong et al.[10] RR =33.53 (2.06-545.50)], reflecting low precision. The pooled success rate supports the efficacy of non-fluoroscopy methods, although some studies showed only marginal differences 96% vs. 97%).[11]

Table 1. Risk of bias

Study ID	Experimental	Comparator	Outcome	D1	D2	D3	D4	D5	Overall
Ackermann et al.[11]	Eptinezumab	Placebo	Monthly migraine days	Low risk	Low risk	Low risk	Low risk	Low risk	Low risk
Kong et al.[10]	Eptinezumab	Placebo	Monthly migraine days	Some concerns	Low risk	Low risk	Some concerns	Low risk	Some concerns
Xu et al.[4]	Eptinezumab	Placebo	Monthly migraine days	Low risk	Low risk	Low risk	Low risk	Low risk	High risk
Ewert et al.[12]	Eptinezumab	Placebo	Monthly migraine days	Some concerns	Low risk	Low risk	High risk	Low risk	

D1: Randomisation process, D2: Deviations from the intended interventions, D3: Missing outcome data, D4: Measurement of the outcome, D5: Selection of the reported result

Table 2. Systematic review table

First author, year	Study design	Origin	Baseline						
			Participants	Age (years)		n		ASD diameter (mm)	
				Fluoroscopy	Non-fluoroscopy	Fluoroscopy	Non-fluoroscopy	Fluoroscopy	Non-fluoroscopy
Ackermann et al.[11]	Retrospective, observational, single-center	University Children's Hospital Zurich, Switzerland	Children undergoing transcatheter ASD closure between 2002 and 2016	6.1 (3.8-10.6)	5.7 (4.1-9.6)	141 female and 97 male (n=238)	103 female and 56 male (n=159)	13.5	12.3
Xu et al.[4]	Retrospective study	Children's Hospital, Zhejiang University School of Medicine, China	Children who underwent percutaneous ASD closure at the hospital between November 2014 and January 2017	62.5 (38.8) months (5.2 years)	71.7 (40.7) months	66 men and 97 female (n=163)	54 men and 76 female (n=130)	9.3 (3.9)	9.9 (4.2)
Kong et al.[10]	Prospective randomized multicenter trial	Fuwai Hospital, Beijing; People's Hospital of Xinjiang Uygur Autonomous Region, Urumqi; Henan Provincial People's Hospital, Zhengzhou, China	Patients presenting with ASD at 3 centers, from July 2018 to September 2019	11 (3-63)	12.5 (2-65)	48	52	10 (5-28)	10 (5-27.9)
Ewert et al.[12]	Retrospective study	Berlin, Germany	All patients with either an ASD of the secundum type or a persistent foramen ovale after presumed paradoxical embolism who were considered suitable for transcatheter closure from July 1998 to May 1999	34 (1-78)	18 (2-66)	131	22	11 (4-26)	9 (6-26)

Table 2. Continued

Comparison	Outcome									
	Successful rate		Median procedure time (min)		Intraprocedural complications (n)		Post-operative complications (n)		Postoperative hospital stay (days)	
	Fluoroscopy	Non-fluoroscopy	Fluoroscopy	Non-fluoroscopy	Fluoroscopy	Non-fluoroscopy	Fluoroscopy	Non-fluoroscopy	Fluoroscopy	Non-fluoroscopy
Ackermann et al.[11] intraprocedural fluoroscopy ± TEE guidance; group 2: TEE guidance alone	96% (n=238)	97% (n=154)	60	34	8	1	8	4	-	-
Xu et al.[4]	100%	100%	28.6 (10.9)	21.5 (14.6)	1	0	21	1	2.9 (0.6)	3.2 (0.6)
Kong et al.[10]	33 (68.75%)	52 (100%)	34	28	8	0	0	0	-	-
Ewert et al.[12]	128/131	22/22	100	88	-	-	-	-	-	-

ASD: Atrial septal defect, PAN: Percutaneous and non-fluoroscopic, TEE: Transoesophageal echocardiography

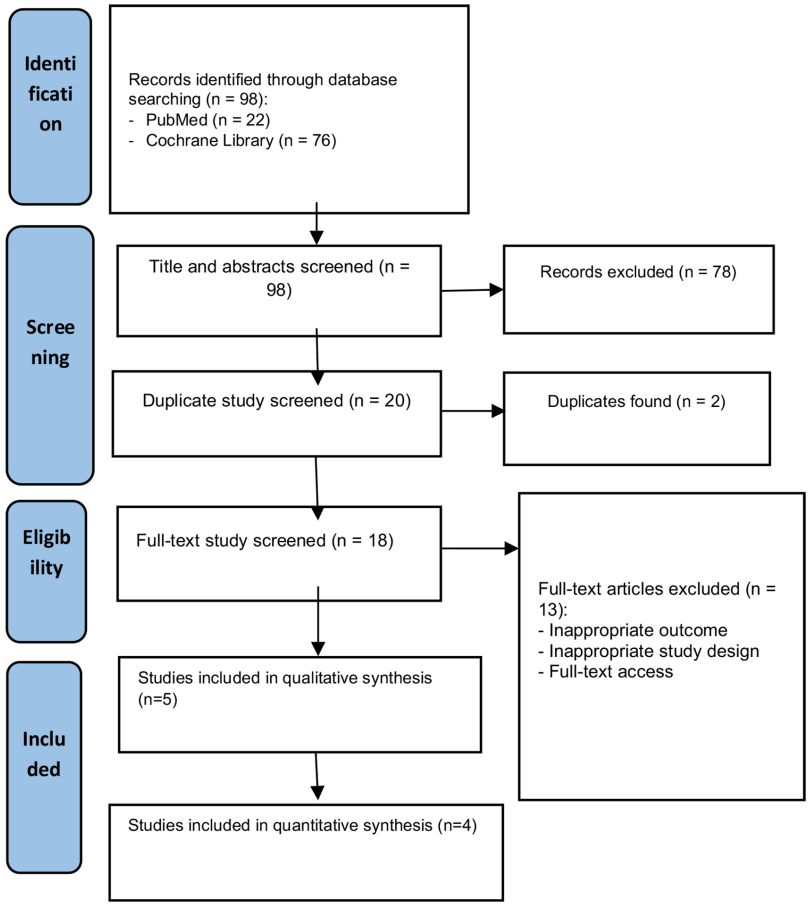

Figure 1. Diagram flow of literature search strategy for this systematic review

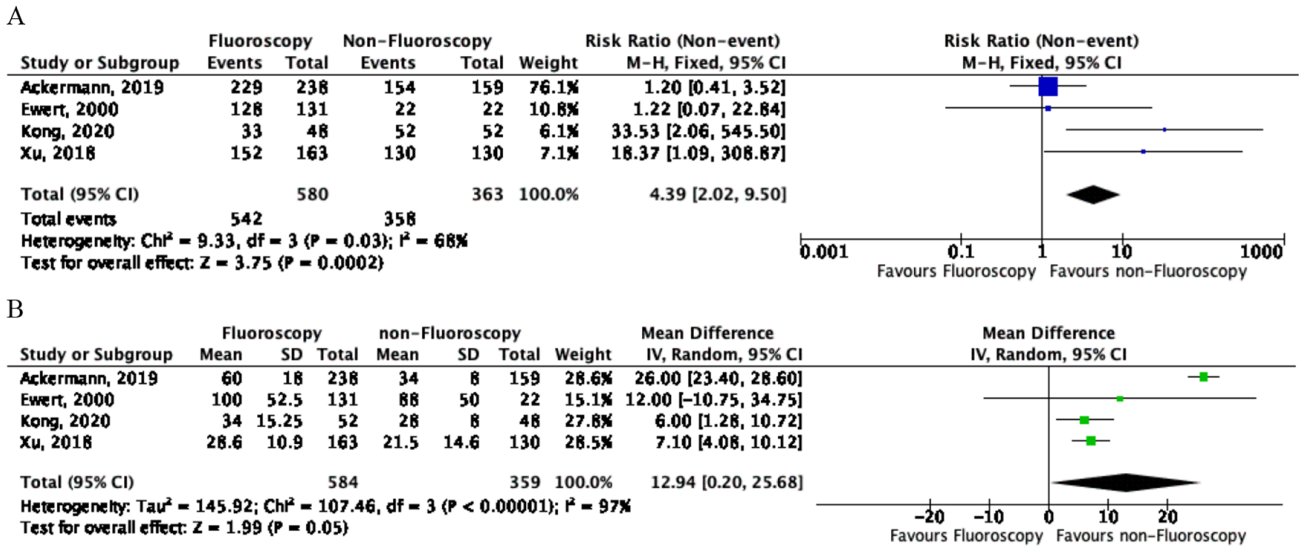

Figure 2. Forest plot of successful event (A) and procedure duration (B) between fluoroscopy *versus* non-fluoroscopy group

CI: Confidence interval

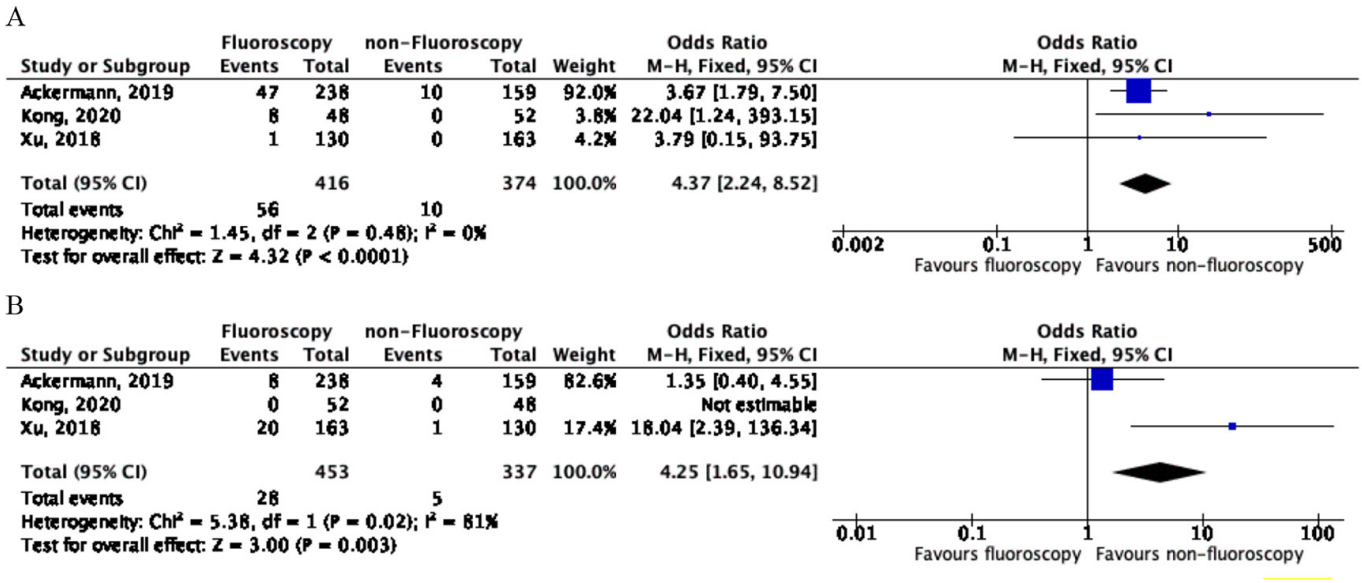

Figure 3. Forest plot of intraprocedural complication (A) and postoperative complications (B) between fluoroscopy *versus* non-fluoroscopy group

CI: Confidence interval

Operative success was determined based on specific criteria: successful passage of the guidewire through ASD into the left atrium (LA), successful entry of the delivery sheath into the LA guided by the guidewire without descending into the right atrium after removal of the guidewire, successful removal of the guidewire from the patient. The definition of procedural success was not standardized across studies, potentially contributing to heterogeneity. Moreover, while the pooled analysis favors non-fluoroscopy, results must be interpreted with caution due to variations in patient selection, imaging modalities (TEE vs. TTE), operator experience, and device types.[12]

The study with the largest number of participants reported a high success rate for transcatheter ASD device closures across both examined groups. Specifically, 229 out of 238 cases (96%) in the fluoroscopy group and 154 out of 159 cases (97%) in the non-fluoroscopy group achieved successful closure ($P = 0.736$). For the nine unsuccessful cases in the fluoroscopy group,

surgery was performed successfully. In the non-fluoroscopy group, secondary fluoroscopy guidance was employed, leading to successful interventional ASD device closure in four out of five of their initially unsuccessful cases (Figure 2A).

The MD of 12.59 indicates that, on average, there is a 12.59 minute difference in procedure time between fluoroscopy-guided and non-fluoroscopy-guided procedures for transcatheter ASD closure. The average duration of procedures for percutaneous ASD closure under fluoroscopic guidance has been documented to vary between 40 and 110 minutes, with variations in how studies define total procedure time.[4,9] Xu et al.[4] showed that the non-fluoroscopy group had a shorter duration [21.5 (14.6) min] than the fluoroscopy group [28.6 (10.9) min], when evaluated from heparinization to removal of the delivery system ($P < 0.001$), with a difference of 7 min (~25%). Procedure durations for fluoroscopy and non-fluoroscopy are summarized in Figure 2B.

An OR of 4.37 indicates a statistically significant association between fluoroscopy guidance and intraprocedural complications in transcatheter ASD closure. This means that patients undergoing fluoroscopy-guided procedures have approximately 4.37 times higher odds of experiencing intraprocedural complications than those undergoing non-fluoroscopy-guided procedures. The CI indicated that this association was relatively precise, ranging from 2.24 to 8.52. Ackermann et al.[11] found intraprocedural complications in 8 cases (3.3%) in the fluoroscopy group, including temporary cardiac rhythm abnormalities, such as transient atrioventricular (AV) dissociation, transient AV block, or non-sustained supraventricular tachycardia, in six cases, and intraprocedural device embolization in two cases. In the non-fluoroscopy group, a male patient underwent Amplatzer septal occluder device embolization due to a defective retroaortic ASD rim. A plot comparing intraprocedural and postoperative complication rates between fluoroscopy and non-fluoroscopy is presented in Figure 3A. This meta-analysis indicates that fluoroscopy-guided transcatheter ASD closure is significantly more likely to result in intraprocedural complications than non-fluoroscopy-guided treatments.

The OR of 3.22, calculated using the Mantel-Haenszel method with fixed effects, indicates that patients undergoing fluoroscopy-guided ASD closure have a 3.22 times higher likelihood of experiencing postoperative complications than those undergoing non-fluoroscopy-guided closure procedures. Major adverse events were categorized as any complications arising within 1 month of the procedures, related to either the devices used or the procedure itself. These complications included but were not limited to death, the necessity of urgent surgery, severe cardiac tamponade requiring drainage or surgical repair, cardiac perforation, hemorrhage, and strokes.[10]

Ewert et al.[12] 12 found that non-fluoroscopy procedures using TEE (88 min) were numerically shorter than with fluoroscopy (100 min); while the success rates differed between the groups, this variation was not considered statistically significant ($P = 0.09$). Several reasons could account for this observed difference. First, TEE provides clear imaging, enabling precise assessment of ASD characteristics such as location, size, and shape, helping to select the most suitable closure device on the first attempt. Second, TEE allows real-time visualization of various components such as steel wires, sheaths, and occlusion devices, facilitating the assessment of residual shunting and the impact of the occlusion device on surrounding structures such as AV valves, pulmonary veins, vena cava, and coronary sinus opening. This capability significantly decreases the procedural time. Third, conventional X-ray equipment may not accurately depict cardiac anatomy, requiring equipment rotation and repeated TEE examinations, thus lengthening procedure duration. In summary, this meta-analysis suggests that fluoroscopy-guided procedures take longer than non-fluoroscopy-guided procedures, with an estimated MD ranging from 1.34 to 23.83 min.

In the study by Xu et al.[4] postoperative fever (temperature above 38 °C) was less common in the non-fluoroscopy group (TEE) than in the fluoroscopy group, with 1 of 130 patients compared to 15 of 163 patients (0.8 versus 9.2%, $P < 0.001$). The rates of other complications were not significantly different between the groups (as shown in Figure 3B). Additionally, no patients in either group experienced postoperative residual shunts, shedding or displacement of the occlusion device, or pericardial effusion.

Our findings support the use of non-fluoroscopy-guided ASD closure, which resulted in improved outcomes and reduced radiation risk. The evidence, while promising, is based on a limited number of studies. The results may not generalize to all populations due to variation in imaging technologies and operator experience. Sensitivity analysis confirmed the robustness of our findings. However, no funnel plot asymmetry was observed, suggesting minimal publication bias.

Study Limitations

The generalizability of these findings is limited by the inclusion of only four studies, which also exhibit moderate heterogeneity among their study populations. Furthermore, a significant drawback is the absence of long-term follow-up data, preventing a comprehensive understanding of long-term outcomes. Lastly, the fact that this analysis was not registered in PROSPERO could raise concerns regarding potential reporting biases.

CONCLUSION

Non-fluoroscopy-guided ASD closure is a promising alternative to fluoroscopy, offering comparable or superior clinical outcomes with lower radiation risk. Larger multicenter studies with standardized definitions and long-term follow-up are needed to confirm these findings.

Footnotes

Authorship Contributions

Surgical and Medical Practices: H.W.P., Concept: K.K., H.W.P., Design: H.W.P., Data Collection or Processing: K.K., Analysis or Interpretation: H.W.P., Literature Search: K.K., Writing: K.K.

Conflict of Interest: No conflict of interest was declared by the authors.

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RESEARCH ARTICLE

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Study of Coronary Artery Disease Severity with HbA1c in Patients with Diabetes Mellitus with SYNTAX Score II - A Prospective Observational Study

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Abstract

Background and Aim: Coronary artery disease (CAD) is a major contributor to mortality in those with diabetes. Chronic hyperglycaemia exacerbates endothelial dysfunction, vascular inflammation, and atherosclerosis, hence worsening the severity of CAD. The SYNTAX score II (SSII) is a common tool for assessing the complexity of CAD and guiding treatment decisions. This study seeks to evaluate the severity of CAD in diabetic patients using the SSII and examining the association between glycated hemoglobin (HbA1c) and additional risk factors.

Materials and Methods: An observational study was conducted at SRM Medical College Hospital and Research Centre, enrolling 121 diabetic patients with angiographically confirmed CAD. SSII was applied to classify patients into low (<22), intermediate (23-32), and high (≥33) risk categories. Logistic regression and chi-square tests were employed to assess the associations between HbA1c levels, duration of diabetes, and severity of CAD.

Results: The average HbA1c was 8.53%±1.68, and the diabetes duration was 7.17±4.64 years. Higher HbA1c levels were significantly associated with severe CAD (P = 0.040), with each 1% increase in HbA1c raising the odds of high-risk SSII by 62.9% [odds ratio (OR) =1.62, P = 0.014]. Prolonged diabetes duration (OR =1.13, P = 0.049) and reduced left ventricular ejection fraction (OR =0.0004, P = 0.019) were also independent predictors.

Conclusion: Elevated HbA1c levels and prolonged diabetes duration are strongly associated with CAD severity in diabetic patients. SSII functions as a valuable instrument for risk stratification and the formulation of treatment plans.

Keywords: Coronary artery disease, diabetes mellitus, SYNTAX score II, HbA1c, cardiovascular risk, glycemic control

INTRODUCTION

The prevalence of coronary artery disease (CAD), particularly in the diabetic population, is an important public health issue worldwide. Diabetes accelerates atherosclerosis by increasing endothelial dysfunction, chronic inflammation, and oxidative stress, resulting in more severe CAD. Diabetes mellitus (DM) substantially elevates cardiovascular risk profiles, while cardiovascular pathologies continue to be the primary cause of global mortality.[1] About 77 million people in India had diabetes in 2019. That number is expected to increase to 134 million by 2045, which would make it one of the countries with the highest diabetes burdens in the world.[2] The prevalence varies regionally, with urban populations and states like Kerala, Tamil Nadu, and Punjab exhibiting higher rates.[3] Given this increasing burden, identifying predictors of CAD severity is essential for improving clinical outcomes.

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Glycated hemoglobin (HbA1c) is a crucial indicator of sustained glycemic regulation and is associated with cardiovascular risk. Increased HbA1c levels facilitate atherosclerosis advancement, plaque instability, and elevated coronary complexity.[4] Uncontrolled diabetes contributes to endothelial dysfunction, systemic inflammation, and a prothrombotic condition, all of which intensify the severity of CAD. Research indicates that a 1% elevation in HbA1c is associated with an approximate 18% increase in cardiovascular risk with people sustaining HbA1c levels over 5.6% exhibit a higher risk for coronary stenosis.[5,6]

The SYNTAX score II (SSII) is an established angiographic tool used to quantify CAD severity by assessing the complexity of coronary lesions. Studies have shown a strong relationship between SYNTAX scores and HbA1c levels, suggesting that more severe coronary lesions are linked to poorer glycemic control.[7] Nonetheless, few studies have investigated the direct correlation between HbA1c levels and the severity of CAD utilizing SSII, particularly in Indian populations, where genetic and lifestyle factors contribute to early-onset and aggressive disease progression. This study aims to assess the severity of CAD in diabetic populations utilizing SSII and examines the association of HbA1c in predicting the complexity of disease.

METHODS

Patients and Study Design

This prospective observational longitudinal study was conducted at SRM Medical College Hospital and Research Centre, Kattankulathur, Chengalpattu, a tertiary healthcare facility. The study aimed to evaluate the association between HbA1c levels and the severity of CAD in diabetic patients. Following approval from the Ethics Committee of the SRM Medical Faculty Hospital and Research Center, (protocol no: SRMIEC-ST0723-790, date: 14.09.2023), the study was conducted over 13 months, from September 2023 to October 2024. The study was conducted in accordance with the principles of the Declaration of Helsinki and Good Clinical Practice guidelines of the International Council for Harmonisation. All participants provided written consent before enrolment.

The sample size was estimated based on the expected effect size in a multivariable logistic regression model, the primary analytic method in this study. Using Cohen's f^2 framework for regression, a medium effect size (f^2=0.15) with up to 10 predictors, an alpha of 0.05, and 80% power requires a minimum of 118 subjects to detect significant associations.[8] Our final sample size of 121, therefore, exceeds this requirement, ensuring adequate power to detect moderate associations between HbA1c, clinical covariates, and CAD severity.

Patients were enrolled consecutively according to predefined inclusion and exclusion criteria. Consecutive sampling was used to minimize selection bias and ensure representativeness of the eligible population. The study included patients with a confirmed diagnosis of DM, HbA1c levels above 6.5%, and angiographically confirmed CAD. Patients with a history of prior coronary revascularization [percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG)], hemoglobinopathies, anemia, recent blood transfusion, or HbA1c below 6.5% were excluded. Inclusion of only angiographically confirmed CAD limits external validity to the diabetic patients with established disease. Informed consent was obtained from all participants before enrolment.

Coronary Angiography and SYNTAX Score II Assessment

All patients underwent coronary angiography to assess CAD severity. The SSII was calculated using dedicated software (version 2.11, www.syntaxscore.com), incorporating clinical and angiographic parameters such as age, renal function, left ventricular ejection fraction (LVEF), and lesion complexity. Patients were stratified into low, intermediate, and high SSII categories using thresholds derived from existing literature. Specifically, tertile-based groupings (≤22, 23-32, ≥33) as used by Serruys et al.[9] were adopted to assess associations with clinical outcomes.

Study Procedure

Patients meeting the inclusion criteria underwent HbA1c measurement before coronary angiography. Following angiographic assessment, the SSII was determined, and treatment recommendations for revascularization (PCI vs. CABG) were formulated based on the severity of CAD.

Statistical Analysis

Comprehensive statistical methodologies were employed to assess the relationship between HbA1c levels and CAD severity as quantified by the SSII. Continuous variables were presented as mean ± standard deviation or median with interquartile range, depending on the distribution characteristics. Categorical variables were represented as proportions or percentages. To find the association between CAD and other risk factors, the chi-square test was used for categorical variables, and the Kruskal-Wallis test was used for continuous variables. Post-hoc pairwise comparisons were performed using the Bonferroni correction. Regression analyses were performed to evaluate the predictive value of HbA1c for CAD severity, adjusting for potential confounding variables including age, gender, renal function, and comorbid conditions. Statistical significance was set at $P < 0.05$.

The scoring system stratifies risk as follows:

- Low risk classification: <22
- Moderate risk classification: 23-32
- High risk classification: ≥33

RESULTS

The study included 121 diabetic patients with CAD, with 91.7% who were above 45 years of age and a male preponderance (65.5%). 28.1% of patients reported smoking, while 33.1% reported using alcohol. Triple vessel disease (TVD) was present in 32.2% of cases. Chronic obstructive pulmonary disease (COPD) was observed in 3.3% of patients, while peripheral vascular disease (PVD) was noted in 2.5% of patients. Systemic hypertension was observed in 81% of the study population (Table 1). Table 2 summarizes the descriptive statistics of the duration of diabetes, HbA1c, lipid profile, renal parameters, and echocardiography.

The SSII classification categorized 77 patients as low risk, 26 as intermediate risk, and 18 as high risk (Table 3). Gender was not significantly associated with CAD severity ($P = 0.373$), while in severe cases, TVD was more prevalent ($P = 0.002$). No significant differences were found in COPD ($P = 0.244$), PVD ($P = 0.148$), or alcohol consumption ($P = 0.237$). Patients exhibiting more severe CAD demonstrated a prolonged duration of DM ($P = 0.004$), elevated HbA1c levels ($P = 0.040$), increased low-density lipoprotein (LDL) cholesterol ($P = 0.043$), and decreased high-density lipoprotein cholesterol (HDL) cholesterol ($P = 0.026$). Creatinine clearance demonstrated a significant decline ($P < 0.001$) and LVEF decreased with the progression of CAD severity ($P = 0.05$).

Post-hoc analysis revealed a significant difference in diabetes duration between low and high-risk groups ($P = 0.008$) and intermediate and high-risk groups ($P = 0.002$); however, the difference between low and intermediate-risk groups was not significant ($P = 0.088$). HbA1c levels were significantly elevated in the high group compared to the low group ($P = 0.005$) and in the intermediate group compared to the high group ($P = 0.001$), but not between the low and intermediate groups ($P = 0.065$). Creatinine clearance was significantly lower in low-risk groups, compared to high-risk groups ($P < 0.001$); and in intermediate groups, compared to high-risk groups ($P = 0.012$); however, no significant difference was observed between low and intermediate risk groups ($P = 0.471$). LVEF was significantly lower in high-risk patients: the comparisons low vs. high ($P = 0.021$) and intermediate vs. high ($P = 0.023$) showed statistical significance. LDL levels were significantly higher in high vs. low ($P = 0.049$) and intermediate vs. high ($P = 0.023$), while HDL levels were significantly lower in high-risk patients ($P = 0.028$ and $P = 0.013$, respectively) (Table 4).

Table 1. Patient characteristics and comorbidities

Category		n (%)
Age group	Less than 45 years	10 (8.3%)
	More than 45 years	111 (91.7%)
Gender	Female	43 (35.54%)
	Male	78 (64.46%)
Smoking	Yes	34 (28.10%)
	No	87 (71.90%)
Alcohol	Yes	40 (33.10%)
	No	81 (66.90%)
TVD	Yes	39 (32.20%)
	No	82 (67.80%)
COPD	Yes	4 (3.30%)
	No	117 (96.70%)
PVD	Yes	3 (2.50%)
	No	118 (97.50%)
SHTN	Yes	98 (81.00%)
	No	23 (19.00%)

TVD: Triple vessel disease, COPD: Chronic obstructive pulmonary disease, PVD: Peripheral vascular disease, SHTN: Systemic hypertension

Table 2. Descriptive statistics of diabetes duration, glycemic control, lipid profile, ejection fraction and renal function parameters

Variable	Mean	Median	SD	Minimum	Maximum
Duration of DM	7.168	5	4.6388	0.7	20
HbA1c	8.529	8.3	1.6868	5.6	13.3
Cholesterol	181.752	172	57.8033	68	349
LDL	128.826	126	43.2446	41	261
HDL	40.421	38	11.9977	18	87
TG	175.884	131	153.7642	30	1349
CrCl (mL/min)	88.752	95	21.3472	15	123
Creatinine	0.931	0.8	0.4577	0.4	4.5
LVEF	0.528	0.52	0.09	0.3	0.67

DM: Diabetes mellitus, LVEF: Left ventricular ejection fraction, CrCl: Creatinine clearance, SD: Standard deviation, LDL: Low-density lipoprotein cholesterol, HDL: High-density lipoprotein cholesterol

Significant predictors of CAD severity, the duration of DM and HbA1c, according to multinomial logistic regression analysis with the low-risk group as the reference category. In the intermediate-risk group, each year's increase in diabetes duration increased the odds of CAD severity by 11.2% [odds ratio (OR) =1.11, $P = 0.049$], and each unit's increase in HbA1c raised the risk by 35.4% (OR =1.35, $P = 0.049$). Creatinine clearance ($P = 0.342$) and LVEF ($P = 0.992$) exhibited no significant predictive value in this group. In the high-risk group, the duration of diabetes was a significant predictor, with each additional year associated with a 1.13 times higher odds of being in the severe CAD group (OR =1.13, $P = 0.049$). Each 1% increase in HbA1c was associated with 1.62 times higher odds of being in the severe CAD group (OR = 1.62, $P = 0.014$). Lower creatinine clearance was a significant predictor of severe CAD (OR =0.95, $P = 0.001$), while reduced LVEF showed a strong association with severe CAD (OR =0.0004, $P = 0.019$) (Table 5).

Table 3. Association between CAD and other risk factors using chi-square test for categorical variables and the Kruskal-Wallis test for continuous variables

Variable		Low (n=77)	Intermediate (n=26)	High (n=18)	*P*-value
Gender	Female	25 (32.5%)	9 (34.6%)	9 (50.0%)	0.373
	Male	52 (67.5%)	17 (65.4%)	9 (50.0%)	
TVD	Yes	18 (23.4%)	9 (34.6%)	12 (66.7%)	0.002
	No	59 (76.6%)	17 (65.4%)	6 (33.3%)	
COPD	Yes	1 (1.3%)	2 (7.7%)	1 (5.6%)	0.244
	No	76 (98.7%)	24 (92.3%)	17 (94.4%)	
PVD	Yes	1 (1.3%)	2 (7.7%)	0 (0%)	0.148
	No	76 (98.7%)	24 (92.3%)	18 (100%)	
Alcohol	Yes	20 (26.0%)	6 (23.1%)	8 (44.4%)	0.237
	No	57 (74.0%)	20 (76.9%)	10 (55.6%)	
Age		58.026±8.19	58.00±8.14	60.72±10.23	0.621
Duration of DM		6.08±3.93	8.35±4.94	10.10±5.52	0.004
HbA1c		7.32±1.48	8.38±1.74	9.19±1.94	0.040
Cholesterol		184.29±56.23	181.42±63.76	171.39±57.70	0.573
LDL		120.70±34.94	134.04±38.47	147.72±40.71	0.043
HDL		40.27±10.76	37.77±14.73	35.56±18.37	0.026
TGL		166.86±106.70	213.42±255.42	160.28±131.46	0.616
CrCl (mL/min)		93.25±18.44	88.81±21.57	69.44±22.94	<0.001
LVEF		0.54±0.09	0.53±0.09	0.48±0.09	0.05

TVD: Triple vessel disease, COPD: Chronic obstructive pulmonary disease, PVD: Peripheral vascular disease, DM: Diabetes mellitus, HbA1c: Glycated hemoglobin, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TGL: Triglycerides, LVEF: Left ventricular ejection fraction, CAD: Coronary artery disease

Table 4. Post-hoc analysis between the CAD and other risk factors

Variable	Comparison groups	W statistic	*P*-value
Duration of DM	Low vs. intermediate	2.98	0.088
	Low vs. high	4.2	0.008
	Intermediate vs. high	1.63	0.002
HbA1c	Low vs. intermediate	3.164	0.065
	Low vs. high	1.24	0.005
	Intermediate vs. high	2.34	0.001
Creatinine clearance (mL/min)	Low vs. intermediate	-1.66	0.471
	Low vs. high	-5.22	<0.001
	Intermediate vs. high	-4.06	0.012

Table 4. Continued

Variable	Comparison groups	W statistic	*P*-value
LVEF	Low vs. intermediate	1.22	0.665
	Low vs. high	3.8	0.021
	Intermediate vs. high	3.6	0.023
LDL	Low vs. intermediate	6.22	0.665
	Low vs. high	4.8	0.049
	Intermediate vs. high	4.6	0.023
HDL	Low vs. intermediate	5.22	0.665
	Low vs. high	5.5	0.028
	Intermediate vs. high	5.7	0.013

CAD: Coronary artery disease, DM: Diabetes mellitus, LVEF: Left ventricular ejection fraction, LDL: Low-density lipoprotein cholesterol, HDL: High-density lipoprotein cholesterol

Table 5. Comparison between the CAD and other risk factors using multinomial logistic regression

CAD severity	Predictor	Estimate	SE	Z	*P*-value	OR (95% CI)
Intermediate	Duration of DM	0.1065	0.0541	1.9675	0.049	1.11(1.00-1.24)
	HbA1c	0.303	0.1538	1.9697	0.049	1.35 (1.00-1.83)
	Creatinine clearance (mL/min)	-0.0119	0.0125	-0.951	0.342	0.99 (0.96-1.01)
	Left ventricular ejection fraction	-0.0256	2.5286	-0.0101	0.992	0.97 (0.69-1.38)
High	Duration of DM	0.1256	0.0638	1.9682	0.049	1.13 (1.00-1.29)
	HbA1c	0.4878	0.1986	2.456	0.014	1.63 (1.10-2.40)
	Creatinine clearance (mL/min)	-0.0447	0.0138	-3.2402	0.001	0.96 (0.93-0.98)
	Left ventricular ejection fraction	-7.7541	3.2934	-2.3545	0.019	0.0004 (0.0000007-0.273)

CAD: Coronary artery disease, DM: Diabetes mellitus, HbA1c: Glycated hemoglobin, LVEF: Left ventricular ejection fraction, SE: Standard error, OR: Odds ratio, CI: Confidence interval

DISCUSSION

This study evaluates the distribution of biochemical, clinical, and demographic parameters across the mild, moderate, and severe levels of CAD severity. The incidence was higher for males in all categories, but there was no significant difference in the gender distribution between groups ($P = 0.373$). Nonetheless, the high-severity group experienced TVD more frequently (66.7%, $P = 0.002$), suggesting that increasing coronary involvement is associated with more severe CAD. There was no statistically significant difference in the prevalence of PVD and COPD across the groups ($P = 0.148$ and $P = 0.244$, respectively), indicating that these comorbidities were equally distributed throughout CAD severity levels. Patients with high severity consumed more alcohol (44.4%), although the disparity was not statistically significant ($P = 0.237$). This implies that while alcohol intake may influence overall health, it may not have a direct impact on CAD severity in this cohort.

Clinical and biochemical parameters demonstrated significant variations among the severity groups. The association between uncontrolled diabetes and deteriorating coronary involvement was further supported by the substantial correlations between higher HbA1c levels, longer duration of diabetes, and greater CAD severity ($P = 0.040$ and 0.004, respectively). Ma et al.[10] emphasized that poor glycemic management and long-term diabetes both significantly contribute to coronary pathology, and they found a strong correlation between higher HbA1c and the severity of CAD. There were also noticeable differences in the lipid profiles, with the high-severity group having lower HDL levels ($P = 0.026$) and higher LDL levels ($P = 0.043$). In line with our results, Achila et al.[11] comparative data from research that included 319 elderly individuals in Asmara, Eritrea, showed a mean total cholesterol of 202.2±40.63 mg/dL and LDL-C of 125.95±33.16 mg/dL. Triglyceride levels were also higher in our sample (175.8 mg/dL) than in their research (129±57.1 mg/dL), suggesting a larger burden of dyslipidaemia in our cohort.

A significant correlation between the severity of CAD and cardiac and renal function was also observed. Creatinine clearance was significantly lower in patients in the high-severity group ($P < 0.001$), suggesting that more severe CAD is associated with deteriorating renal function. In the same way, the high-severity group's LVEF was significantly lower ($P = 0.050$), suggesting that those with advanced CAD had diminished cardiac function.

Key determinants of the severity of CAD were identified using multinomial logistic regression analysis. Higher HbA1c levels (OR =1.35, P = 0.049) and longer diabetes duration (OR =1.11, P = 0.049) were significant predictors in the intermediate-severity group. Duration of DM was associated with higher odds of severe CAD (OR =1.13, 95% CI: 1.00-1.28, P = 0.049), though the association was borderline, and higher HbA1c (OR =1.62, P = 0.014) continued to be a significant predictor of high-severity CAD. The negative impact of uncontrolled diabetes on the development of CAD was highlighted by Khan et al.[12], who discovered a strong correlation between severe coronary involvement and HbA1c values over 7.5%, with a mean HbA1c of 10.23±2.58%, which is similar to our study, in which the mean HbA1c was 8.53%. Similar findings were reported by Ma et al.[10] and Jiao et al.[13], confirming the association between elevated HbA1c and severe CAD in diabetic populations.

Each 1% increase in HbA1c was associated with higher odds of belonging to the severe CAD group according to our study (OR =1.62, P = 0.014). These findings align with studies from Northeastern India, where HbA1c levels above 5.1% independently predicted SYNTAX scores exceeding 22 [OR =1.6, 95% confidence interval (CI): 1.04-2.46], reinforcing the strong relationship between uncontrolled diabetes and complex coronary disease.[13,14] Furthermore, data analysis revealed, that each 1% increase in HbA1c corresponded to a 15% elevated risk of severe coronary heart disease (OR =1.14).

In addition to glycemic control, reduced creatinine clearance and lower LVEF were also associated with severe CAD. In multinomial logistic regression, lower creatinine clearance was significantly associated with severe CAD (OR =0.96, 95% CI: 0.93-0.98, P = 0.001), indicating that each 1 mL/min decline in creatinine clearance corresponded to approximately 4% higher odds of being in the severe CAD group. This emphasizes the predictive significance of renal function in assessing the severity of CAD, as even minor declines in kidney function significantly impact cardiovascular outcomes. Pereg et al.[14] discovered that a drop of 10 mL/min in creatinine clearance was associated with a 4.77-fold increased risk of developing CAD, particularly in young, healthy males without diabetes or cardiovascular disease. Furthermore, LVEF was found to be a strong predictor of CAD severity, with lower ejection fractions correlating with more extensive coronary disease. Our findings that cardiac dysfunction is a crucial indicator of advanced coronary disease, were supported by Dorbala et al.[15], who found a clear correlation between decreasing LVEF and increasing CAD complexity.

In comparison with previous research, this study highlights strong associations between worsening CAD and reduced LVEF, uncontrolled diabetes, impaired kidney function, and longer duration of diabetes. This study clarifies the association between diabetes, glycemic control, renal function, lipid profile, and the severity of CAD. Future longitudinal multicenter research, including genetic and lifestyle factors, could provide more data on how CAD develops in diabetic populations.

Study Limitations

As a prospective observational study without long-term follow-up, this study design limits the ability to establish causality or assess temporal changes between variables. As it is a single-center study, the possibility of selection bias cannot be fully excluded. Our study excludes well-controlled diabetics, which restricts the generalizability of the findings. Furthermore, the observed relationships may be influenced by the absence of information on relevant confounders, including nutrition, exercise, and genetic susceptibility, necessitating further thorough investigation.

CONCLUSION

Prolonged duration of illness and uncontrolled diabetes contribute to increased severity of CAD, with the latter significantly influencing severity. HbA1c is strongly associated with angiographic severity of CAD. The link between metabolic, renal, and cardiac dysfunction emphasizes the necessity for a comprehensive risk management strategy. Effective glycemic control, cholesterol modulation, and renal function monitoring are critical in lowering the CAD burden in diabetic patients, as they stress the need for early and personalized therapeutic interventions.

Ethics

Ethics Committee Approval: The study was obtained from the Ethics Committee of SRM Faculty of Medicine Hospital and Research Center (protocol no: SRMIEC-ST0723-790, date: September 14, 2023).

Informed Consent: All participants provided written informed consent.

Footnotes

Authorship Contributions

Surgical and Medical Practices: K.V., P.R., V.T.A., Concept: K.V., A.S.B., P.R., V.T.A., R.J.D., Design: A.S.B., P.R., V.T.A., R.J.D., Data Collection or Processing: K.V., A.S.B., R.J.D., Analysis or Interpretation: K.V., P.R., V.T.A., R.J.D., Literature Search: P.R., V.T.A., R.J.D., Writing: K.V., A.S.B., P.R., V.T.A., R.J.D.

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RESEARCH ARTICLE

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Evaluation of the Relationship Between HATCH Score and SYNTAX Score in Patients Undergoing Coronary Angiography

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Abstract

Background and Aim: Originally developed to predict the progression of paroxysmal atrial fibrillation, the hypertension (1 point), age >75 years (1 point), transient ischemic attack or stroke (2 points), chronic obstructive pulmonary disease (1 point), heart failure (2 points) (HATCH) score has recently been explored as a broader prognostic tool in cardiovascular medicine. This study investigates the relationship between the HATCH score and the Synergy between PCI with Taxus and Cardiac Surgery (SYNTAX) score, a well-established measure of coronary artery disease (CAD) complexity, among patients with chronic coronary syndrome (CCS).

Materials and Methods: We retrospectively analyzed data from 235 patients who underwent coronary angiography for suspected CAD between January 2023 and May 2024. Patients were categorized into two groups based on their SYNTAX scores: low (≤22) and intermediate-high (>22). Demographic, clinical, and laboratory parameters—including the HATCH score—were compared between groups. Univariate analyses and Firth's penalized logistic regression were performed to identify independent predictors of higher SYNTAX scores. Receiver operating characteristic (ROC) analysis was used to evaluate the discriminative performance of the HATCH score.

Results: Patients with intermediate-high SYNTAX scores were significantly older, were more likely to have hypertension, heart failure, prior percutaneous coronary intervention, and renal dysfunction, and had lower left ventricular ejection fraction than those with low SYNTAX scores. Among all evaluated variables, the HATCH score emerged as the strongest independent predictor of intermediate-high coronary complexity [odds ratio: 3.815; 95% confidence interval (CI): 2.656-4.233; $P < 0.001$]. ROC analysis demonstrated good discriminative capacity, with an area under the curve of 0.805 (95% CI: 0.740-0.870; $P < 0.001$). A HATCH score cut-off of ≥2 yielded a specificity of 87% and a sensitivity of 72%.

Conclusion: The HATCH score, based on accessible clinical parameters, is independently associated with CAD complexity in CCS patients. Its simplicity and high specificity make it a useful tool for early risk stratification in clinical practice. Prospective multicenter studies are needed to validate its prognostic value and clinical utility.

Keywords: HATCH score, SYNTAX score, chronic coronary syndrome, coronary artery disease

INTRODUCTION

The term "chronic coronary syndromes (CCS)" refers to a framework introduced by the European Society of Cardiology in 2019 to replace the term "stable coronary artery disease (CAD)". This conceptual shift acknowledges the evolving and multifaceted nature of CAD, which is now understood not as a static entity but as a progressive and dynamic condition. CCS encompasses various clinical scenarios, including individuals presenting with suspected CAD who exhibit exertional angina and/or dyspnoea that remain stable over time.[1] Within the

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broader population of individuals diagnosed with coronary heart disease (CHD), approximately 30-36% are hospitalized for chronic manifestations of CHD.[2]

Invasive diagnostic strategies remain pivotal in assessing cardiovascular disease, as they provide comprehensive and irreplaceable information regarding both coronary anatomy and the physiological significance of coronary lesions. Despite advances in non-invasive imaging modalities, such approaches are not currently considered viable alternatives, because they do not match the diagnostic accuracy and therapeutic guidance provided by invasive evaluation.[1,3]

Multivessel CAD (MV-CAD) refers to the coexistence of hemodynamically significant atherosclerotic lesions in two or more major epicardial coronary arteries. This condition typically involves substantial luminal narrowing, most often defined as ≥50% stenosis, affecting epicardial coronary arteries, such as the left anterior descending artery, the right coronary artery, or the left circumflex artery. The presence of MV-CAD across various clinical presentations, including acute and CCS, is of critical importance for therapeutic decision-making and survival outcomes.[4-6] Given the paramount clinical significance of MV-CAD in cardiology practice, the Synergy between percutaneous coronary intervention (PCI) with Taxus and Cardiac Surgery (SYNTAX) score was developed to objectively quantify the anatomical complexity of CAD.[7]

de Vos et al.[8] introduced the hypertension (1 point), age >75 years (1 point), transient ischemic attack or stroke (2 points), chronic obstructive pulmonary disease (1 point), heart failure (2 points) (HATCH) score, a clinically practical and easily applied risk stratification tool developed to estimate the likelihood that atrial fibrillation will progress from paroxysmal to persistent. The score is an acronym derived from five key clinical variables: hypertension (HTN), age >75 years, transient ischemic attack or stroke, chronic obstructive pulmonary disease (COPD), and heart failure (HF).[8] In addition to its established role in forecasting the progression of atrial fibrillation, the HATCH score has emerged as a significant prognostic indicator of adverse clinical outcomes, including mortality and hospitalization, in patients with HF, regardless of the presence of atrial fibrillation.[9] Building upon the growing evidence supporting the HATCH score as a potential prognostic marker beyond its original application in atrial fibrillation, we designed our study to explore its relevance within the context of CCS. Specifically, our objective was to examine the relationship between the HATCH score and the SYNTAX score. We evaluated whether the HATCH score, composed of readily obtainable clinical parameters, could reflect the burden and severity of coronary atherosclerosis, as represented by the SYNTAX score, in patients with CCS.

METHODS

Study Population

This retrospective study included consecutive patients admitted to our tertiary care center between January 2023 and May 2024. Eligible participants were adults aged 18 years or older who underwent coronary angiography due to clinical symptoms indicative of ischemic heart disease, abnormal findings on non-invasive stress testing suggestive of underlying CAD, or both. The exclusion criteria for this study included acute coronary syndrome on presentation, a history of coronary artery bypass graft surgery, angiographic findings showing of less than 50% coronary artery stenosis or angiographically normal coronary arteries, and lack of clinical or laboratory data.

Baseline clinical, demographic, and laboratory data were collected from the hospital's electronic health system. The study population was divided into two groups based on SYNTAX score: low and intermediate-high. Comparative analyses were conducted to evaluate differences in clinical and laboratory parameters, as well as the HATCH score, between the two groups.

Ethical approval for this study was obtained from the Ethics Committee of University of Health Science Türkiye, Bakırköy Dr. Sadi Konuk Training and Research Hospital our tertiary care institution (approval number: 2025-15-07, date: 20.08.2025). The study was conducted in full accordance with the ethical principles outlined in the Declaration of Helsinki. Informed consent was obtained from all individual participants prior to their inclusion in the study.

Definitions

CCS was defined as a clinical spectrum that includes patients with suspected CAD who have "stable" anginal symptoms and/or dyspnoea, as well as asymptomatic and symptomatic patients more than 1 year after initial diagnosis or revascularization.[1]

The SYNTAX score, a validated measure of coronary anatomical complexity, was calculated from coronary angiograms acquired during hospitalization using the official SYNTAX score online calculator. Two cardiologists, blinded to the patients' clinical information, independently performed the scoring to minimize observer bias.[10]

The HATCH score was calculated by assigning 1 point for HTN, 1 point for age >75 years, 2 points for a history of stroke or transient ischemic attack, 1 point for COPD, and 2 points for HF. The total score was calculated as the sum of these components.[9]

Left ventricular ejection fraction (LVEF) was assessed using the modified Simpson's method by transthoracic echocardiography performed prior to coronary angiography.[11]

Statistical Analysis

The normality of the distributions of all continuous variables was assessed using the Kolmogorov-Smirnov test. Variables with a normal distribution were reported as mean ± standard deviation, and comparisons between groups were performed using the independent samples t-test. Non-normally distributed variables were summarized as median [interquartile range (IQR)] and compared using the Mann-Whitney U test. Categorical variables were summarized as frequencies and percentages and compared using the chi-square test or Fisher's exact test when appropriate. Univariable logistic regression was performed to identify potential predictors of an intermediate-to-high SYNTAX score. Variables that reached statistical significance in the univariable analysis were subsequently included in Firth's penalized logistic regression to reduce small-sample bias, avoid model overfitting, and identify independent predictors. Multicollinearity was assessed using the variance inflation factor, calculated from the corresponding standard logistic regression models. The correlation between the HATCH and SYNTAX scores was further examined using Spearman's rank correlation analysis. A receiver operating characteristic (ROC) curve analysis was performed to assess the predictive value of the HATCH score for SYNTAX score categories. A post-hoc power analysis was performed to assess the ability of the study to detect differences in HATCH score and in diabetes mellitus (DM) prevalence between patients with SYNTAX scores ≤22 and those with scores >22. The HATCH score differed significantly between the SYNTAX >22 and ≤22 groups [1.00 (0.00-1.00) vs 2.00 (1.00-2.00), $P < 0.001$], with an achieved statistical power of 0.92, indicating sufficient power to detect a true difference between groups. Although the prevalence of DM was higher in the SYNTAX >22 group than in the ≤22 group (55.6% vs. 43.1%), this difference did not reach statistical significance ($P = 0.107$). The post-hoc power analysis for DM revealed a low statistical power of 0.364, suggesting that the non-significant result may reflect an inadequate sample size rather than the absence of a true association. A *P*-value <0.05 was considered statistically significant. All statistical analyses were performed using SPSS software (version 25.0; IBM Corp., Armonk, NY, USA) and R software (version 4.5.0; R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

A total of 235 patients were included in the study, of whom 181 (77.0%) had a SYNTAX score ≤22 (low SYNTAX group) and 54 (23.0%) had a SYNTAX score >22 (intermediate-high SYNTAX group). The median age was significantly higher in the intermediate-high SYNTAX group compared with the low SYNTAX group (68.5 vs. 61.0 years, respectively; $P < 0.001$). There were no statistically significant differences between the groups in sex distribution or body mass index. The prevalence of HTN was significantly higher among patients in the intermediate-high SYNTAX group compared with those in the low SYNTAX group (79.6% vs. 53.6%; $P = 0.001$). In addition, HF and a history of PCI were more prevalent in the intermediate-high SYNTAX group. In contrast, no statistically significant differences were observed between groups with respect to smoking status, DM, cerebrovascular disease, COPD, or statin use. Laboratory parameters, including hemoglobin, platelet and white blood cell counts, glucose, triglycerides, LDL cholesterol, uric acid, albumin, and C-reactive protein levels, were comparable between the two groups. However, urea and creatinine levels were significantly higher in the intermediate-high SYNTAX group than in the low SYNTAX group. LVEF was significantly lower in the intermediate-high SYNTAX group (50.0% vs. 60.0%; $P < 0.001$). The median HATCH score was 1.00 (IQR: 0.00-1.00) in the low SYNTAX group and 2.00 (IQR: 1.00-2.00) in the intermediate-high SYNTAX group, with a statistically significant difference between the groups ($P < 0.001$). A significant positive correlation was observed between the SYNTAX score and the HATCH score (r=0.633, $P < 0.001$), as determined by Spearman's correlation analysis. Baseline demographic, clinical, and laboratory parameters are summarized in Table 1.

Univariate logistic regression analysis identified several clinical and laboratory variables significantly associated with advanced SYNTAX scores (>22), indicative of greater CAD complexity. Specifically, older age, HTN, HF, history of PCI, and elevated serum urea and creatinine levels were significantly associated with a higher SYNTAX score. Conversely, LVEF exhibited a statistically significant inverse relationship with SYNTAX score, such that increased LVEF was associated with a lower SYNTAX score. Notably, the HATCH score was a robust predictor of intermediate-to-high SYNTAX scores, demonstrating a statistically significant association [odds ratio (OR): 4.298; 95% confidence interval (CI): 2.676-7.543; $P < 0.001$], indicating that higher HATCH scores substantially increase the likelihood of intermediate-to-high SYNTAX scores (Table 2).

Firth's penalized logistic regression was used to identify the independent predictors of intermediate-to-high SYNTAX scores. In model A, which included serum creatinine, prior PCI, LVEF, and the HATCH score, the HATCH score emerged as a strong and statistically significant predictor. The HATCH score was the strongest predictor of an intermediate-high SYNTAX score (OR: 3.815; 95% CI: 2.656-4.233; $P < 0.001$). Serum creatinine, prior PCI and LVEF were identified as additional independent predictors of an intermediate-to-high SYNTAX score. In model B, age, HTN, HF, prior PCI, creatinine, and LVEF were identified as independent predictors of intermediate-to-high SYNTAX scores (Table 3).

ROC curve analysis demonstrated that the HATCH score exhibited good discriminative capacity for predicting intermediate-high SYNTAX scores, as evidenced by an area under the curve of 0.805 ($P < 0.001$; 95% CI: 0.740-0.870). With a cut-off of 2, the HATCH score demonstrated a high specificity of 87%, highlighting its ability to accurately identify patients without advanced CAD. Sensitivity was moderate at 72%, reflecting a conservative detection approach with limited ability to identify all patients with complex coronary lesions (Figure 1). These metrics collectively reinforce the HATCH score's potential as a pragmatic instrument for nuanced risk stratification in CAD.

DISCUSSION

In this retrospective study of patients with CCS, we assessed the severity and extent of CAD using the SYNTAX score and examined its association with the HATCH score. The principal finding was that the HATCH score demonstrated a strong, independent association with intermediate-high SYNTAX scores, suggesting its potential utility in identifying patients at greater risk for anatomically complex CAD. In addition to the HATCH score, age, HTN, LVEF, creatinine, prior PCI and HF were independent predictors of advanced SYNTAX scores in our analysis. The HATCH score, age, HTN, creatinine, prior PCI and HF were positively associated with advanced SYNTAX scores, whereas LVEF demonstrated an inverse relationship with the complexity of CAD.

Table 1: Comparison of clinical, laboratory parameters and HATCH score between low and intermediate-high SYNTAX score groups

Variable	Overall, n=235 (IQR)	Low SYNTAX group, n=181 (IQR)	Intermediate-high SYNTAX, n=54 (IQR)	*P*-value
Age (years)	69.00 (63.00-69.00)	61.00 (55.00-68.00)	68.50 (62.00-73.00)	<0.001
Female gender	67 (28.5%)	49 (27.1%)	18 (33.3%)	0.371
BMI	28.45±4.80	28.40±4.63	28.64±5.38	0.896
Smoking	153 (65.1%)	121 (66.9%)	32 (59.3%)	0.304
DM	102 (43.4%)	78 (43.1%)	24 (44.4%)	0.861
HTN	140 (59.6%)	97 (53.6%)	43 (79.6%)	0.001
Stroke or TIA	7 (3.0%)	3 (1.7%)	4 (7.4%)	0.051
COPD	22 (9.4%)	14 (7.7%)	8 (14.8%)	0.179
HF	15 (6.4)	7 (3.9)	8 (14.8)	0.004
Prior PCI	66 (28.1)	42 (23.2)	24 (44.4)	0.002
ACEI/ARB	145 (617)	109 (60.2)	36 (66.7)	0.397
Statin	216 (91.9)	164 (90.6)	52 (96.3)	0.178
SYNTAX score	14.00 (10.00–22.00)	13.00 (9.00-16.75)	26.50 (24.50-35.00)	<0.001
Hemoglobin (g/dL)	13.53±1.71	13.63±1.69	13.20±1.73	0.132
Platelet (×10³/µL)	239.00 (201.00-288.50)	240.00 (204.00-287.00)	236.00 (191.50-293.25)	0.576
Neutrophil (×10³/µL)	4.74 (3.63-6.07)	4.74 (3.57-6.07)	4.78 (3.93-6.42)	0.532
Lymphocyte (×10³/µL)	2.01 (1.59-2.58)	2.09 (1.61-2.64)	1.93 (1.40-2.48)	0.107
Glucose (mg/dL)	111.90 (97.00-147.00)	110.50 (97.15-144.00)	114.95 (96.20-165.75)	0.717
Triglyceride (mg/dL)	158.00 (116.00-223.00)	161.50 (116.00-227.75)	149.50 (115.75-213.75)	0.879
LDL (mg/dL)	97.00 (74.00-134.62)	98.00 (74.00-137.00)	96.00 (79.00-122.50)	0.546
Urea (mg/dL)	33.50 (27.60-41.80)	32.70 (26.70-40.00)	39.30 (31.95-48.70)	0.001
Creatinine (mg/dL)	0.90 (0.78-1.06)	0.88 (0.77-1.05)	0.96 (0.79-1.29)	0.024
Uric acid	5.26 (4.35-6.34)	5.20 (4.27-6.27)	5.60 (4.71-6.65)	0.062
Albumin (g/L)	45.00 (42.10-47.10)	45.15 (42.17-47.17)	44.70 (42.00-46.90)	0.138
CRP (mg/L)	3.20 (1.60-7.25)	3.00 (1.40-7.05)	4.50 (2.00-8.10)	0.195
LVEF (%)	60.00 (50.00-60.00)	60.00 (55.00-60.00)	50.00 (40.00-60.00)	<0.001
HATCH score	1.00 (0.00-1.00)	1.00 (0.00-1.00)	2.00 (1.00-2.00)	<0.001

ACEI: Angiotensin-converting enzyme inhibitors, ARB: Angiotensin II receptor blockers, BMI: Body mass index, COPD: Chronic obstructive pulmonary disease, CRP: C-reactive protein, DM: Diabetes mellitus, HATCH: Hypertension (1 point), age >75 years (1 point), transient ischemic attack or stroke (2 points), chronic obstructive pulmonary disease (1 point), heart failure (2 points), HF: Heart failure, HTN: Hypertension, LDL: Low-density lipoprotein, LVEF: Left ventricular ejection fraction, IQR: Interquartile range, PCI: Percutaneous coronary intervention, SYNTAX: Synergy between PCI with Taxus and Cardiac Surgery, TIA: Transient ischemic attack

Table 2: Univariable logistic regression analysis for intermediate-high SYNTAX score

Variable	OR	95% CI	*P*-value
Age	1.093	1.049-1.139	<0.001
HTN	3.385	1.641-6.981	0.001
HF	4.323	1.490-12.542	0.007
Prior PCI	2.455	1.294-4.658	0.006
Urea	1.032	1.010-1.055	0.004
Creatinine	1.427	1.232-2.064	0.049
LVEF	0.907	0.870-0.945	<0.001
HATCH score	4.298	2.676-7.543	<0.001

CI: Confidence interval, HATCH: Hypertension (1 point), age >75 years (1 point), transient ischemic attack or stroke (2 points), chronic obstructive pulmonary disease (1 point), heart failure (2 points), HF: Heart failure, HTN: Hypertension, LVEF: Left ventricular ejection fraction, OR: Odds ratio, PCI: Percutaneous coronary intervention

Table 3: Firth's penalized logistic regression analysis for determing of independent predictors of intermediate-high SYNTAX score

Variable	OR	95% CI	*P*-value	VIF
Model A				
Creatinine	1.332	1.010-1.966	0.044	1.02
Prior PCI	2.568	1.989-6.667	0.043	1.04
LVEF	0.880	0.854-0.996	0.001	1.50
HATCH score	3.815	2.656-4.233	<0.001	1.53
Model B				
Age	1.096	1.045-1.149	<0.001	1.06
HTN	3.421	1.437-8.147	0.005	1.06
HF	2.505	1.168-4.652	0.028	1.03
Prior PCI	2.613	1.340-4.720	0.008	1.04
Creatinine	1.537	1.012-2.334	0.044	1.02
LVEF	0.887	0.844-0.932	<0.001	1.03

CI: Confidence interval, HATCH: Hypertension (1 point), age >75 years (1 point), transient ischemic attack or stroke (2 points), chronic obstructive pulmonary disease (1 point), heart failure (2 points), HF: Heart failure, HTN: Hypertension, LVEF: Left ventricular ejection fraction, OR: Odds ratio, PCI: Percutaneous coronary intervention, VIF: Variance inflation factor

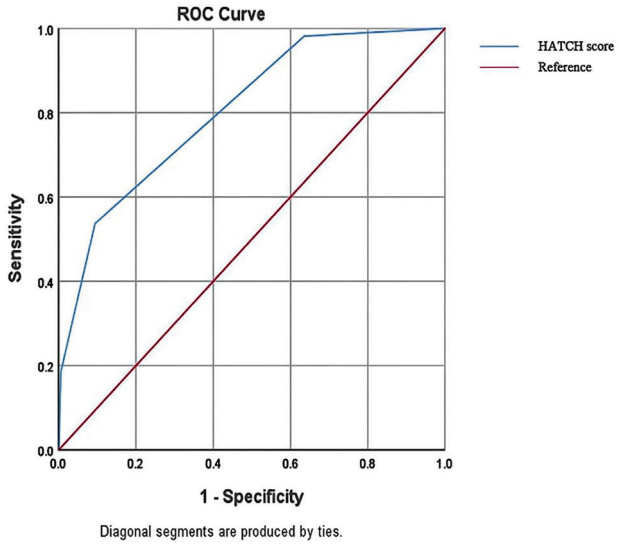

Figure 1: Receiver operating characteristic (ROC) curve analysis of HATCH score for predicting intermediate-high SYNTAX score

AUC: Area under the curve, CI: Confidence interval, HATCH: Hypertension (1 point), age >75 years (1 point), transient ischemic attack or stroke (2 points), chronic obstructive pulmonary disease (1 point), heart failure (2 points), SYNTAX: Synergy between PCI with Taxus and Cardiac Surgery

MV-CAD constitutes a significant clinical challenge requiring thorough evaluation in contemporary cardiology practice. Its presence markedly influences both procedural success in interventional cardiology and patients' long-term prognosis. Sorajja et al.[12] reported that among patients presenting with acute myocardial infarction, procedural success rates tended to decline incrementally in those with two- and three-vessel CAD compared with those with single-vessel involvement, although this trend did not reach statistical significance. In the same study, mortality rates increased incrementally in patients with two- and three-vessel disease compared with those with single-vessel disease; this difference was statistically significant.[12] The detrimental effect of MV-CAD on clinical outcomes extends beyond the setting of acute myocardial infarction. In the study conducted by Lopes et al.[13] MV-CAD was identified as an independent predictor of mortality in patients with stable CAD; its presence was associated with a 1.9- to 3.1-fold increase in long-term mortality risk.

Given the substantial clinical implications of MV-CAD, the identification of its associated risk factors has become increasingly important. Identified risk factors for the development of MV-CAD encompass a range of demographic, clinical, and metabolic variables. These include age, male sex, HTN, DM, lipid metabolism disorders, chronic kidney disease, and a history of myocardial infarction.[14] In our study, age and HTN were identified as independent predictors of more-advanced CAD, defined by an intermediate-high SYNTAX score.

Beyond the well-established traditional cardiovascular risk factors, COPD has increasingly been recognized as a meaningful contributor to the burden of CAD. COPD is no longer viewed solely as a pulmonary condition; emerging evidence points to a significant association between COPD and MV-CAD. This association may arise not only from the shared risk factor, tobacco smoking, but also from systemic inflammation and oxidative stress that characterize both conditions and critically contribute to the progression of vascular disease.

Consistent with expectations, accumulating evidence suggests a significant association between COPD and MV-CAD.[15-18]

Ischemic stroke is a clinical manifestation within the spectrum of atherosclerotic cardiovascular disease. Owing to their common atherosclerotic pathophysiology and overlapping risk factors, a bidirectional relationship between CAD and ischemic stroke has been well established in the literature. Notably, individuals with a prior history of stroke exhibit a significantly increased risk of developing CAD, while patients already diagnosed with CAD are likewise at increased risk of subsequent ischemic stroke.[19,20] Furthermore, findings from a registry study have highlighted a robust association between MV-CAD and ischemic stroke, underscoring the clinical relevance of both conditions, which share common pathophysiological mechanisms and overlapping risk factors.[20]

An additional clinical parameter that merits thorough consideration in the context of MV-CAD is left ventricular function. Accumulating evidence from multiple studies indicates that patients with a decline in left ventricular systolic performance, as quantified by reduced LVEF, are more likely to have MV-CAD.[21,22] Indeed, in our study, an inverse relationship was observed between LVEF and intermediate-high SYNTAX scores. Specifically, as LVEF decreased, the likelihood of detecting more complex CAD increased. This association may be explained by the involvement of a larger myocardial territory subjected to ischemia, which consequently leads to systolic dysfunction. The relationship between MV-CAD and left ventricular systolic function extends beyond LVEF. Indeed, Bruno et al.[23] demonstrated that complete revascularization in patients with MV-CAD significantly reduced hospitalizations for HF.

A comprehensive evaluation of the current scientific evidence reveals that several key clinical conditions—including advanced age, HTN, cerebrovascular accident, COPD, and HF—are significantly associated with the presence of MV-CAD. By integrating these five clinical variables, the HATCH score provides a comprehensive yet straightforward method for predicting the severity and anatomical complexity of MV-CAD. The HATCH score's simplicity, cost-effectiveness, and reliance on routinely available clinical parameters render it a valuable tool for broad implementation in clinical practice. By consolidating multiple high-risk comorbid conditions into a unified scoring system, it is possible to facilitate the early identification of individuals at increased risk for anatomically complex CAD. This enables clinicians to adopt a more timely, evidence-based, and individualized management approach, thereby potentially improving both short- and long-term cardiovascular outcomes.

Although the HATCH score was originally developed to estimate the likelihood of progression from paroxysmal to sustained atrial fibrillation, several of its constituent variables are also relevant to CAD. Specifically, HTN and advanced age—both well-established major risk factors for CAD—and a history of transient ischemic attacks or cerebrovascular events, which represent clinical manifestations within the broader cardiovascular disease spectrum, have been associated with the presence and severity of CAD. Moreover, these factors may contribute to vascular aging, further linking the HATCH score components to coronary complexity. This pathophysiological overlap provides a biologically plausible rationale for investigating the potential utility of the HATCH score in the assessment of coronary complexity, although rigorous validation in this context remains necessary.

Study Limitations

This study has several limitations that warrant careful consideration. Firstly, its retrospective, observational, and single-center architecture intrinsically constrains external generalizability, as the results may not extrapolate to broader, heterogeneous populations or disparate clinical milieus. Second, although the HATCH score incorporates several well-established cardiovascular risk factors, it does not account for other potentially influential contributors to coronary complexity, such as inflammatory status, genetic predisposition, or lifestyle-related variables. Third, the unequal distribution across the SYNTAX groups (low SYNTAX group: 181 patients; intermediate-high SYNTAX group: 54 patients) may have limited the statistical power to detect differences in the smaller group and may have affected the robustness of the multivariable analysis. Additionally, the study lacked an independent validation cohort, and no sensitivity analyses were conducted to assess the robustness and reproducibility of the findings. Another limitation is that internal validation techniques, including cross-validation or bootstrapping, were not employed, limiting the assessment of model stability. Moreover, potential selection bias may have influenced the findings, as the study population was derived from a single-center retrospective cohort. Lastly, the absence of long-term follow-up data precludes any assessment of the prognostic implications of the HATCH score, particularly in relation to adverse cardiovascular outcomes.

CONCLUSION

In conclusion, this investigation elucidates that the HATCH score has a strong and independent association with intermediate-high SYNTAX scores, underscoring its utility as a pragmatic and integrative tool for appraising the anatomical complexity of CAD in patients with CCS. Beyond conventional determinants such as advancing age or HTN, the HATCH score integrates multiple clinical risk factors into a single accessible metric, thereby enhancing the clinician's capacity to identify individuals at increased risk of multivessel involvement at an early stage. Its simplicity, cost-effectiveness, and reliance

on routinely procured clinical data support its incorporation into cardiovascular assessment paradigms. Nonetheless, the generalizability of these findings necessitates corroboration in large, prospective, multicenter cohorts, alongside exploration of their prognostic implications for long-term clinical trajectories.

Ethics

Ethics Committee Approval: Ethical approval for this study was obtained from the Ethics Committee of University of Health Science Türkiye, Bakırköy Dr. Sadi Konuk Training and Research Hospital our tertiary care institution (approval number: 2025-15-07, date: 20.08.2025). The study was conducted in full accordance with the ethical principles outlined in the Declaration of Helsinki.

Informed Consent: Informed consent was obtained from all individual participants prior to their inclusion in the study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: E.A., C.Y., Concept: E.A., C.Y., Design: E.A., C.Y., Data Collection or Processing: E.A., C.Y., Analysis or Interpretation: E.A., C.Y., Literature Search: E.A., C.Y., Writing: E.A.

Conflict of Interest: No conflict of interest was declared by the authors.

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RESEARCH ARTICLE

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Short-term Outcome of Patients with Acute Myocardial Infarction and SCAI-classified Cardiogenic Shock: An Egyptian Registry

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Abstract

Background and Aim: Acute myocardial infarction complicated by cardiogenic shock (AMI-CS) is associated with high morbidity and mortality. The Society for Cardiovascular Angiography and Interventions (SCAI) shock classification provides a structured approach to risk stratification. This study examines the predictors of in-hospital and 30-day mortality among AMI-CS patients using the SCAI staging classification.

Materials and Methods: A prospective cohort study was conducted on 150 patients admitted with AMI-CS at Department of Cardiology, Ain Shams University Hospitals from November 2023 to August 2024. Patients were categorized into SCAI stages (A to E) at presentation and reassessed 24 hours later. Demographic, clinical, biochemical, and hemodynamic parameters were collected.

Results: At presentation, 35.3% of patients were in stage A, 4% in stage B, 54.7% in stage C, 4.7% in stage D, and 1.3% in stage E. Overall in-hospital and 30-day mortalities were 28.7% and 37.3%, respectively. Higher SCAI stages correlated with increased mortality ($P < 0.001$). Independent predictors of mortality included a Sequential Organ Failure Assessment score greater than 5 [odds ratio (OR) =8.17, $P < 0.001$], an APACHE score greater than 7 (OR =3.71, $P = 0.008$), and a serum creatinine level greater than 1.53 mg/dL (OR =5.37, $P = 0.005$). The SCAI score at 24 hours demonstrated superior predictive accuracy for in-hospital mortality (area under the curve =0.889).

Conclusions: SCAI staging is a valuable prognostic tool for MI patients with CS. Reassessment at 24 hours enhances mortality prediction, emphasizing the importance of dynamic risk stratification.

Keywords: Myocardial infarction, cardiogenic shock, SCAI classification, mortality prediction, risk stratification

INTRODUCTION

Acute myocardial infarction (AMI) results from the partial or complete interruption of blood flow to a region of the myocardium. It remains a leading cause of morbidity and mortality worldwide, affecting over three million people annually.[1] It is diagnosed by a rise and/or fall in cardiac troponins above the 99th percentile, accompanied by at least one of the following: ischemic symptoms, segmental wall motion abnormalities on echocardiography, new left bundle branch block (LBBB) or pathological Q waves on Electrocardiogram (ECG), or the presence of an intracoronary thrombus detected by coronary angiography or at autopsy.[2] Multiple modifiable and non-modifiable risk factors play a role in AMI.[3]

Cardiogenic shock (CS) is a leading cause of morbidity and mortality among patients with myocardial infarction, characterized by critical end-organ hypoperfusion and hypoxia resulting from primary cardiac dysfunction.[4] It is defined by hemodynamic criteria, including systolic blood pressure (SBP)

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<90 mmHg for more than 30 minutes (without hypovolemia or requirement for vasopressors), a reduced cardiac index (less than 1.8 L/min/m² without IV support or less than 2.2 L/min/m² with IV support), and elevated left ventricular filling pressures (pulmonary capillary wedge pressure >18 mmHg).[5]

Clinical signs of organ hypoperfusion include cold extremities, reduced urine output, and altered mental status. Serum lactate is used to assess microcirculatory impairment. CS remains the most severe complication of AMI, contributing to nearly 50% of associated deaths.[6] To improve risk stratification, the Society for Cardiovascular Angiography and Interventions (SCAI) has proposed a five-stage classification, based on physical examination, biochemical markers, and hemodynamic parameters: stage A "at risk", stage B "beginning", stage C "classic", stage D "deteriorating", and stage E "extremis".[7]

Cardiogenic Shock Risk Scores

In the cardiac care unit (CCU), various risk scores are employed to assess the severity of CS and predict patient outcomes, including mortality and morbidity. These scores include:

1. The SCAI staging system (SCAI) classifies CS into five stages, from at-risk (stage A) to end-stage (stage E), to guide treatment and assess severity.
2. Sequential organ failure assessment (SOFA) score measures the extent of organ dysfunction by evaluating the respiratory, coagulation, hepatic, cardiovascular, renal, and neurological systems. Higher scores indicate more severe organ failure and a greater risk.
3. Acute physiology and chronic health evaluation II (APACHE II) score: Estimates mortality risk based on acute physiological variables, age, and chronic health conditions, incorporating factors such as temperature, blood pressure, and heart rate.
4. Simplified acute physiology score II predicts mortality risk based on acute physiological measurements and chronic health status, including heart rate, blood pressure, and laboratory results.
5. The CS prognosis score is specifically designed for CS and evaluates prognosis and severity by integrating clinical variables to guide treatment decisions.

Aim of The Work

This study aims to determine the predictors of in-hospital and 30-day outcomes in patients with AMI with CS (AMI-CS), based on the SCAI Shock classification.

METHODS

Design and Population

This prospective cohort study (a prospective observational study without intervention and therefore not eligible for registration in a clinical trial registry) included 150 AMI-CS patients, categorized according to the SCAI classification (applied prospectively at presentation: A to E), in the Cardiology Department of Department of Cardiology, Ain Shams University Hospitals from November 2023 to August 2024. The study protocol was approved by the Scientific and Ethical Committee of Ain Shams University Faculty of Medicine (approval number: FMASU MS161/2024, date: 03.03.2024). All participants provided informed consent; privacy and confidentiality were ensured.

CONSORT Flow Diagram

Eligibility Criteria

The study included patients with AMI who were classified according to the SCAI shock staging system.[2,8] Patients were diagnosed with AMI according to the Fourth Universal Definition of Myocardial Infarction, which requires a rise and/or fall of cardiac troponin levels with at least one value above the 99th percentile upper reference limit, together with at least one of the following: ischemic symptoms; new ischemic ECG changes (such as ST-segment elevation or depression, or new LBBB); imaging evidence of new loss of viable myocardium or of new regional wall motion abnormalities; or identification of intracoronary thrombus via angiography.[2] Exclusion criteria included patients who died before reaching the coronary care unit when sufficient clinical and lab data could not be collected to classify them according to the SCAI staging system.

All Patients Were Subjected to Various Assessments, Including

1-Detailed history taking including: Detailed history taking included demographics (age, gender), comorbid conditions, current medications, history of myocardial infarction and heart failure, previous percutaneous coronary intervention (PCI) or coronary artery bypass grafting, presence of chronic kidney disease (CKD) or prior dialysis, timing of chest pain onset, and occurrence of out-of-hospital cardiac arrest.

The Charlson comorbidity index was used to quantify comorbidities and predict mortality risk.[9]

2-Clinical assessments upon admission included a special emphasis on measuring arterial blood pressure [systolic, diastolic, and mean arterial pressure (MAP), calculated as diastolic blood pressure +1/3 pulse pressure], heart rate, respiratory rate, and oxygen saturation. Chest and cardiac auscultation were performed to assess pulmonary and cardiac

status. Additionally, the Glasgow coma scale (GCS) was used to evaluate the neurological function.[10]

3-Twelve-lead ECG: ECGs were recorded at baseline using a CM 300 A (Comen, China) machine. ST-segment elevation was defined according to the fourth universal definition of myocardial infarction as an ST-segment elevation at the J-point in two or more contiguous leads, with the following thresholds—≥1 mm (0.1 mV) in all leads except V2-V3; in leads V2-V3, elevation of ≥2 mm (0.2 mV) in men aged ≥40 years, ≥2.5 mm (0.25 mV) in men aged <40 years, or ≥1.5 mm (0.15 mV) in women, regardless of age, was considered significant. T wave abnormalities were also assessed according to standard guidelines. Based on these findings, patients were classified as ST-elevation myocardial infarction (STEMI) or non-STMEI (NSTEMI).[2]

4-Laboratory investigations: Random blood sugar at admission, complete blood count, kidney function tests (blood urea nitrogen, creatinine), alanine aminotransferase, HbA1c, creatine kinase total, creatine kinase myocardial band, troponins, arterial pH, bicarbonate, serum lactate, and glomerular filtration rate (GFR) calculated using the CKD epidemiology collaboration equation.

5-Coronary angiography and revascularization: This procedure was performed by experienced interventional cardiologists, and comprehensive procedural details were documented, including balloon predilatation, thrombus aspiration, intracoronary GPIIb/IIIa inhibitors, and the choice of angioplasty alone, stenting alone, or angioplasty followed by stenting.

6-Thrombolysis in myocardial infarction (TIMI) flow grade: Assessed pre- and post-PCI (for the culprit vessel), classifying coronary perfusion from grade 0 (no flow) to grade 3 (complete perfusion).[11]

7-Myocardial blush grade: Recorded pre- and post-PCI (for the culprit vessel), ranging from grade 0 (no blush or persistent staining) to grade 3 (normal blush).[12]

8-Revascularization strategy: Complete versus culprit-only revascularization, with full revascularization defined as an intervention on all significantly diseased vessels with stenosis more than 70%.

9-Additional interventions: The need for mechanical ventilation before or during PCI, the use of mechanical circulatory support (e.g., intra-aortic balloon pump), and the presence of single- or multivessel disease.

10-SCAI shock stages: Classified based on hemodynamic stability, presence of hypoperfusion, and need for inotropic or mechanical support. Class A (at risk) included hemodynamically stable patients who had a large AMI and no signs of hypoperfusion. Class B (beginning CS) included patients with relative hypotension or tachycardia but without hypoperfusion or the need for vasopressors. Class C (classic CS) consisted of patients with hypotension (SBP ≤90 mmHg or MAP ≤60 mmHg) and hypoperfusion requiring therapy. Class D (deteriorating CS) included patients whose condition worsened despite treatment, as indicated by increasing lactate levels or escalating vasoactive drug requirements. Class E (extremis) included patients with prolonged cardiac arrest requiring cardiopulmonary resuscitation or extracorporeal membrane oxygenation (ECMO). Additionally, an A-modifier was applied to patients presenting with cardiac arrest. Hypotension and tachycardia were assessed within the first hour of CCU admission.[13]

11-Severity of illness assessment: The in-hospital severity of illness was evaluated using APACHE II and SOFA scores, both predictive of mortality in intensive care unit settings and validated for CCU patients to estimate short- and long-term mortality risk. The APACHE II score was calculated by summing the acute physiology score (based on 12 variables), age points (ranging from 0 for individuals under 44 years to 6 for those 75 years or older), and chronic health points, resulting in a total score ranging from 0 to 71. Severe acute kidney injury was defined as a doubling of serum creatinine from baseline (the lowest known value), an increase in serum creatinine to greater than 4.0 mg/dL, or initiation of new dialysis; patients on prior dialysis were excluded.

12-Echocardiographic assessment: General electric S7 machine using an M4S matrix sector array probe having a frequency of 2.5 mega Hertz. Evaluation included assessment of left ventricular ejection fraction (LVEF) using the biplane Simpson's method from apical four- and two-chamber views; measurement of left ventricular dimensions and volumes; visual assessment of segmental wall motion abnormalities by an experienced cardiologist, scored according to a standardized 17-segment model with each segment classified as normal, hypokinetic, akinetic, or dyskinetic; and evaluation of the presence of mechanical complications.

Interobserver Variability

To ensure that the assessments in our study were consistent and reliable, two experienced cardiologists independently reviewed key measurements, including:

- SCAI shock stages at presentation and after 24 hours
- TIMI flow grade
- Myocardial blush grade
- LVEF by echocardiography
-

If they disagreed, they reviewed the case together to reach a final decision. We used simple percentage agreement and standard measures such as Cohen's kappa and the intraclass correlation coefficient to measure how well they agreed. Agreement was considered good if the score exceeded 0.7.

Sample Size

Using the power analysis and sample size 11 program for sample size calculation with a 95% confidence interval and a 10% margin of error, it was estimated that a sample size of 95 patients with AMI-CS was needed to detect an expected in-hospital mortality rate of 44.73%. Assuming a 10% dropout rate, a sample size of at least 105 patients with AMI-CS was required. In our study, we enrolled 150 patients, which constituted a larger cohort than the minimum estimated to assess mortality frequency. This larger sample size enhances the reliability of our regression analyses.

Handling Missing Data

Missing data were reviewed for all variables. Cases with missing key outcome data were excluded from analysis. For minor missing clinical or lab values (<5%), a complete-case analysis was performed without imputation.

Statistical Analysis

Data were collected, coded, reviewed, and entered into IBM SPSS version 20 for analysis. Categorical variables were summarized as frequencies and percentages, while continuous variables were reported as mean ± standard deviation for normally distributed data and as median with interquartile range for non-normally distributed data. A 95% confidence interval and a 5% margin of error were applied, with statistical significance defined as a *P*-value ≤0.05. Both univariate and multivariate logistic regression analyses were conducted to identify predictors of mortality. Receiver operating characteristic (ROC) curves were used to evaluate SCAI shock stages at presentation and at 24 hours after admission.

RESULTS

In this study, we aimed to evaluate the predictive value of the SCAI shock classification system in patients presenting to our center with AMI complicated by CS in an Egyptian population. Additionally, we sought to assess the role of other clinical parameters in predicting both in-hospital and 30-day mortality outcomes. Our analysis included 150 patients over a 6-month period who were classified according to the SCAI shock stages at presentation and were re-evaluated after 24 hours. We also examined several factors, including the incidence of out-of-hospital cardiac arrest (OHCA), the need for mechanical ventilation, and the impact of serum lactate levels. Of the 150 patients enrolled in our study, 127 underwent coronary intervention, either single- or multivessel, and 23 did not undergo intervention, either because they experienced cardiac arrest before reaching the cath-lab or for procedural reasons.

The study included 150 AMI-CS patients; males accounted for 72% and females for 28%. The mean age was 59±12 years, with a range of 26 to 83 years. The detailed characteristics of the study population are shown in Table 1.

Patients in SCAI stages D and E exhibited significantly worse clinical and biochemical parameters than those in stages A and B. The admission oxygen saturation, GCS, SBP, MAP, and lactate levels differed significantly ($P < 0.001$), indicating greater physiological deterioration. As shown in Table 1.

Advanced SCAI stages were associated with higher creatinine levels ($P = 0.018$), lower baseline GFR ($P < 0.001$), and elevated troponin levels ($P < 0.001$), indicating worsening organ perfusion and myocardial damage. Mechanical ventilation ($P < 0.001$) and OHCA ($P < 0.001$) were strongly associated with advanced SCAI stages, indicating critical illness. TIMI flow grade ($P < 0.001$) and myocardial blush grade ($P = 0.029$) showed significant differences, with lower TIMI flow and poorer myocardial perfusion in severe cases. Additionally, multivessel interventions ($P = 0.002$) were more frequent in SCAI stages D and E, reflecting the presence of complex coronary disease. These findings confirm that higher SCAI stages correlate with severe clinical deterioration, worse hemodynamics, and increased mortality risk, reinforcing the prognostic value of SCAI classification in patients with acute cardiac conditions as shown in Table 1.

SCAI staging showed a significant shift over 24 hours ($P < 0.001$). The proportion of patients in stage A increased from 35.3% to 43.3%, while stage C cases decreased from 54.7% to 30%. Notably, stage B cases increased from 4% to 16.7%, and stage E cases rose from 1.3% to 4.7%, indicating dynamic clinical progression as shown in Table 2.

Among patients who died during hospitalization, 55.8% had initially presented in SCAI stage C, 14.0% in stage D, and 4.7% in stage E. In contrast, among survivors, the majority (54.2%) were in stage C, with only 0.9% in stage D and none in stage E at presentation ($P < 0.001$). These percentages reflect the distribution of SCAI stages at presentation among deceased and surviving patients, not the mortality rate within each stage. Mortality rates per stage are presented separately for clarity as shown in Table 3 and Figure 1.

Table 1: Characteristics of different SCAI stages at presentation						
Variable	**A (n=53)**	**B (n=6)**	**C (n=82)**	**D (n=7)**	**E (n=2)**	***P*-value**
Gender						
Female	14 (26.4%)	1 (16.7%)	23 (28%)	3 (42.9%)	1 (50%)	0.792
Male	39 (73.6%)	5 (83.3%)	59 (72%)	4 (57.1%)	1 (50%)	
Age, years	59.17±10.9	54.83±14.51	59.63±11.65	61.71±18.54	35.5±9.19	0.061
SO_2, %	94.75±3.18	96.33±0.82	92.82±4.12	84.14±5.05	82.5±3.54	<0.001*
GCS	14.68±1.4	15±0	14.27±1.39	8±2.31	4.5±2.12	<0.001*
Heart rate, bpm	90±16.7	93.33±17.51	94.48±26.1	109.29±46.41	85±63.64	0.371
SBP, mmHg	104.15±14.73	88.33±17.22	79.27±9.91	72.86±4.88	60±0	<0.001*
MAP, mmHg	91.79±13.87	75.83±16.56	68.29±9.27	63.57±3.78	52.5±3.54	<0.001*
Myocardial infarction type						
STEMI	44 (83%)	4 (66.7%)	66 (80.5%)	7 (100%)	2 (100%)	0.542
NSTEMI	9 (17%)	2 (33.3%)	16 (19.5%)	0 (0%)	0 (0%)	
CCI	3 (2-4)	2 (1-4)	4 (3-6)	4 (0-6)	2 (1-3)	0.020*
Prior PCI or CABG	14 (26.4%)	1 (16.7%)	26 (31.7%)	1 (14.3%)	0 (0%)	0.656
Heart failure	8 (15.1%)	0 (0%)	16 (19.5%)	1 (14.3%)	0 (0%)	0.699
Previous MI	12 (22.6%)	0 (0%)	22 (26.8%)	1 (14.3%)	0 (0%)	0.504
CKD	6 (11.3%)	1 (16.7%)	14 (17.1%)	2 (28.6%)	0 (0%)	0.706
Hypertension	18 (34%)	3 (50%)	41 (50%)	4 (57.1%)	1 (50%)	0.415
Diabetes	25 (47.2%)	1 (16.7%)	51 (62.2%)	4 (57.1%)	1 (50%)	0.158
Smoking	33 (62.3%)	5 (83.3%)	55 (67.1%)	3 (42.9%)	1 (50%)	0.574
SOFA score	3 (2-6)	3.5 (3-4)	6.5 (5-7)	10.5 (10-12)	12.5 (11-14)	<0.001*
APACHE score	3 (3-7)	2 (1-7)	7 (3-12)	32.5 (12-35)	51.5 (30-73)	<0.001*
Lactate (peak)	2.6±2.13	2.0±0.68	4.76±2.35	8.27±2.22	6.25±1.06	<0.001*
HCO_3	23.18±4.66	23.8±3.25	18.29±3.61	13.75±3.9	15.5±3.54	<0.001*
pH	7.49±0.07	7.42±0.03	7.32±0.1	7.22±0.1	7.17±0.1	<0.001*
Creatinine	1.08 (0.8-1.29)	1.06 (0.8-1.2)	1.17 (0.96-1.5)	1.84 (1.44-2.5)	1.45 (0.8-2.1)	0.018*
Troponin	50000 (3796-50000)	1422.5 (30-5980)	1228 (20.3-10680)	8869.5 (1598-30000)	27500 (5000-50000)	<0.001*
CK-MB	86 (37-170)	41 (20-69)	70.5 (39-164)	94 (67-278)	119.5 (53-186)	0.496
CK total	825 (330-1657)	177.5 (150-339)	645 (209-1788)	425 (333-950)	574.5 (149-1000)	0.267
Baseline GFR	78 (63-98)	92.5 (47-105)	66.5 (43-93)	35 (25-40)	75.5 (43-108)	<0.001*
Ventilator	4 (81.1%)	6 (100%)	53 (64.6%)	0 (0%)	0 (0%)	<0.001*
EF, %	40.3±9.01	44.0±12.6	38.0±9.65	35.7±5.35	37.5±3.54	0.344
OHCA	**No** 53 (100.0%)	6 (100.0%)	72 (87.8%)	1 (14.3%)	2 (100.0%)	<0.001*
	Yes 0 (0%)	0 (0%)	10 (12.2%)	6 (85.7%)	0 (0%)	
Coronary intervention						
No intervention	7 (13.2%)	2 (33.3%)	8 (9.8%)	5 (71.4%)	1 (50.0%)	0.002*
Single vessel	35 (66.0%)	4 (66.7%)	54 (65.9%)	2 (28.6%)	1 (50.0%)	
More than one vessel	11 (20.8%)	0 (0.0%)	20 (24.4%)	0 (0.0%)	0 (0.0%)	
No. of stents	1 (1-2)	1 (0-2)	1 (1-2)	2 (2-2)	1 (1-1)	0.731
TIMI flow						
Zero	6 (11.3%)	1 (16.7%)	4 (4.9%)	5 (71.4%)	1 (50.0%)	<0.001*
I	2 (3.8%)	0 (0.0%)	6 (7.3%)	0 (0.0%)	0 (0.0%)	
II	10 (18.9%)	1 (16.7%)	28 (34.1%)	1 (14.3%)	0 (0.0%)	
III	35 (66.0%)	4 (66.7%)	44 (53.7%)	1 (14.3%)	1 (50.0%)	

Table 1: Continued

Variable	A (n=53)	B (n=6)	C (n=82)	D (n=7)	E (n=2)	*P*-value
Myocardial blush						
Grade 0	5 (9.6%)	1 (16.7%)	2 (2.5%)	0 (0.0%)	0 (0.0%)	
Grade I	2 (3.8%)	0 (0.0%)	5 (6.3%)	1 (50.0%)	0 (0.0%)	0.029*
Grade II	10 (19.2%)	1 (16.7%)	36 (45.0%)	0 (0.0%)	0 (0.0%)	
Grade III	35 (67.3%)	4 (66.7%)	37 (46.3%)	1 (50.0%)	1 (100.0%)	

Data were presented as Mean ± standard deviation, median (interquartile range), n (%), *: Statistically significant *P*-value as $P < 0.05$, CCI: Charlson comorbidity index, SO_2: Oxygen saturation, GCS: Glasgow coma scale, SBP: Systolic blood pressure, MAP: Mean arterial pressure, STEMI: ST-elevation myocardial infarction, NSTEMI: Non-ST-elevation myocardial infarction, PCI: Percutaneous coronary intervention, CABG: Coronary artery bypass grafting, MI: Myocardial Infarction, CKD: Chronic kidney disease, SOFA: Sequential organ failure assessment, APACHE: Acute physiology and chronic health evaluation, HCO_3: Bicarbonate, PH: Potential of Hydrogen (blood pH), CK-MB: Creatine kinase myocardial band, GFR: Glomerular filtration rate, EF: Ejection fraction, OHCA: Out-of-hospital cardiac arrest, TIMI: Thrombolysis in myocardial infarction, HCO_3: Bicarbonate

The table shows a clear clinical deterioration across SCAI shock stages A to E, with significant declines in oxygen saturation, blood pressure, GCS, and pH, and significant increases in lactate, severity scores (SOFA, APACHE), troponin, and creatinine (all $P < 0.05$). Advanced stages were associated with worse perfusion (lower TIMI flow and reduced myocardial blush), more frequent OHCA, and fewer coronary interventions. Demographics, comorbidities, and MI type did not differ significantly

Table 2: SCAI staging at presentation and after 24 hours among the studied patients

		At presentation	After 24 hours	*P*-value
SCAI staging	**A**	53 (35.3%)	65 (43.3%)	<0.001*
	B	6 (4%)	25 (16.7%)	
	C	82 (54.7%)	45 (30%)	
	D	7 (4.7%)	8 (5.3%)	
	E	2 (1.3%)	7 (4.7%)	

*: Statistically significant *P*-value as $P < 0.05$, SCAI: Society for cardiovascular angiography and interventions

Table 2 shows significant changes in SCAI staging over 24 hours ($P < 0.001$), with an overall shift toward less severe stages. The proportion of patients in stage A increased from 35.3% to 43.3%; in stage B, it rose from 4% to 16.7%; and in stage C, it decreased from 54.7% to 30%

Table 3: Relation of in-hospital and 30-day mortality with SCAI staging at presentation and after 24 hours among the studied patients

		In hospital mortality		*P*-value
		Alive (n=107)	**Died (n=43)**	
SCAI staging at presentation	**A**	42 (39.3%)	11 (25.6%)	<0.001*
	B	6 (5.6%)	0 (0%)	
	C	58 (54.2%)	24 (55.8%)	
	D	1 (0.9%)	6 (14%)	
	E	0 (0%)	2 (4.7%)	
24-hour SCAI staging	**A**	62 (57.9%)	3 (7%)	<0.001*
	B	24 (22.4%)	1 (2.3%)	
	C	21 (19.6%)	24 (55.8%)	
	D	0 (0%)	8 (18.6%)	
	E	0 (0%)	7 (16.3%)	
		30 days mortality		
		Alive (n=94)	**Died (n=56)**	
SCAI staging at presentation	**A**	39 (41.5%)	14 (25%)	0.006
	B	5 (5.3%)	1 (1.8%)	
	C	49 (52.1%)	33 (58.9%)	
	D	1 (1.1%)	6 (10.7%)	
	E	0 (0%)	2 (3.6%)	

Table 3: Continued

		In hospital mortality		*P*-value
		Alive (n=107)	Died (n=43)	
24-hour SCAI staging at hours	A	57 (60.6%)	8 (14.3%)	<0.001*
	B	22 (23.4%)	3 (5.4%)	
	C	15 (16%)	30 (53.6%)	
	D	0 (0%)	8 (14.3%)	
	E	0 (0%)	7 (12.5%)	

*: Statistically significant *P*-value as $P < 0.05$, SCAI: Society for cardiovascular angiography and interventions, n: Number

Table 3 shows that higher SCAI stages are strongly associated with increased in-hospital and 30-day mortality rates. At presentation, most deaths occurred in stages C-E, with significantly fewer in A and B ($P < 0.001$). After 24 hours, mortality was highest in stages C-E, while nearly all survivors were in stages A or B

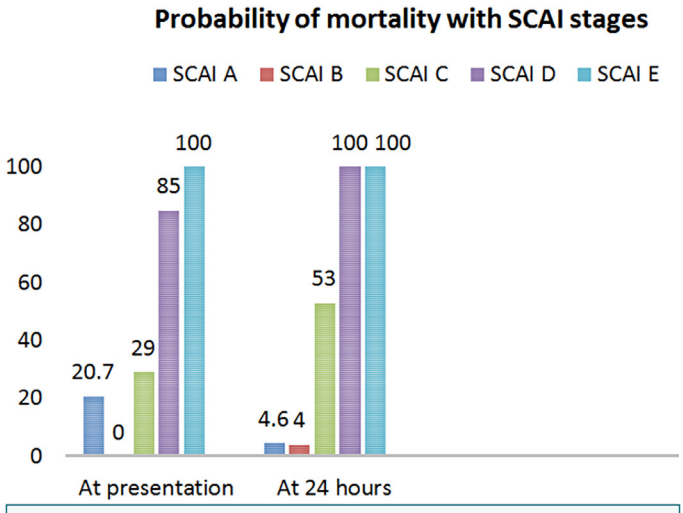

Figure 1: Probability of mortality with advancing SCAI stages at presentation and at 24 hours

SCAI: Society for Cardiovascular Angiography and Interventions

Univariate logistic regression identified several significant predictors of in-hospital mortality, including SOFA score >5 ($P < 0.001$), APACHE score >7 ($P < 0.001$), GCS ≤13 [odds ratio (OR) =10.023, $P < 0.001$), and creatinine >1.53 mg/dL ($P < 0.001$)]. In the multivariate analysis, SOFA score >5 ($P < 0.001$), APACHE score >7 ($P = 0.008$), and creatinine >1.53 mg/dL ($P = 0.005$) remained independent predictors of mortality, indicating their strong prognostic value. Ventilator use showed a trend towards significance (OR =3.083, $P = 0.053$), while other factors lost significance after adjustment as shown in Table 4.

Our data also showed no statistically significant differences in in-hospital or 30-day mortality between genders as shown in Table 5.

Our data also showed no statistically significant difference between single-vessel and complete revascularisation with respect to in-hospital and 30-day mortality Table 6.

The SCAI staging demonstrates an improvement in predictive accuracy over time for both in-hospital and 30-day mortality. At presentation, a cut-off score of >2 yields an area under the curve (AUC) of 0.631 for 30-day mortality and 0.637 for in-hospital mortality, with sensitivities of 73.21% and 74.42% and specificities of 46.81% and 44.86% for 30-day and in-hospital mortality, respectively. While initial predictive ability is moderate, at 24 hours the AUC improves significantly to 0.843 for 30-day mortality and to 0.889 for in-hospital mortality. Sensitivity increases to 80.36% and 90.7%, while specificity increases to 84.04% and 80.37%, respectively. Correspondingly, positive predictive values increased from 45.1% to 75.0% (30-day) and from 35.2% to 65.0% (in-hospital), while negative predictive values increased from 74.6% to 87.8% (30-day) and from 81.4% to 95.6% (in-hospital), highlighting the SCAI score at 24 hours as a more reliable predictor of mortality Figure 2 (A-B) and Table 7.

DISCUSSION

The SCAI shock classification categorizes acute CS into stages A (at risk) through E (extremis). While primarily designed for acute presentations, the SCAI classification also aids in identifying patients at risk of developing CS, particularly those with AMI.[14] Our study aims to validate the SCAI classification for predicting in-hospital and 30-day mortality among AMI patients with CS, while examining demographic and procedural factors that influence short-term outcomes.

Our findings indicate that 35.3% of patients were classified as SCAI stage A at presentation, increasing to 43.3% after 24 hours. Only three deaths occurred in this group, suggesting a relatively favourable prognosis consistent with Baran et al.'s[15] findings. In contrast, patients in stage C or worse exhibited significantly increased mortality, particularly with worsening SCAI stage over 24 hours, further supporting the classification's predictive value for CS severity and outcomes.

Our study found that 35.3% of patients were in stage A, 4% in stage B, 54.7% in stage C, 4.7% in stage D, and 1.3% in stage E at presentation; this differs from Baran et al.[15] who reported no stage A patients and a higher proportion in advanced stages (D and E). These discrepancies may reflect variations in patient demographics, classification criteria, or disease severity across different study populations.

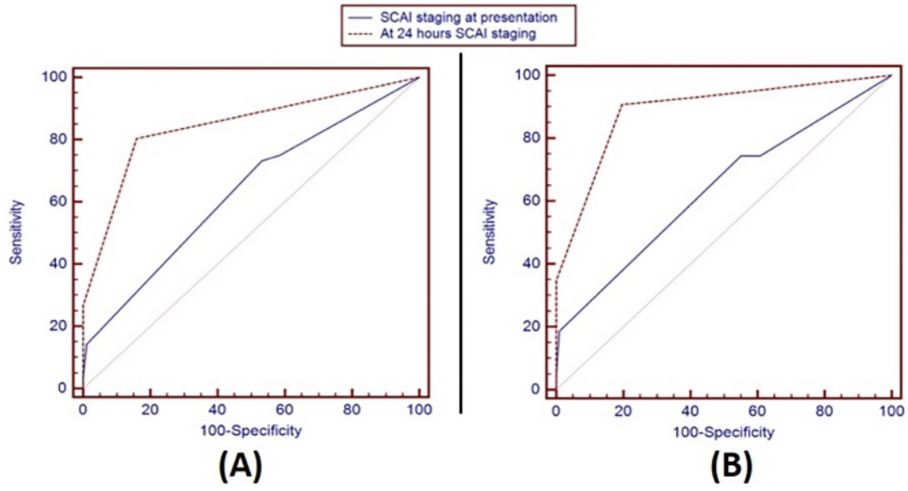

Figure 2: ROC curves assessing SCAI staging at presentation and 24 hours for predicting (A) in-hospital, (B) and 30-day mortality among the studied patients

SCAI: Society for Cardiovascular Angiography and Interventions

Table 4: Univariate and multivariate logistic regression analysis to assess factors associated with the occurrence of in-hospital mortality among the studied patients

	Univariate				Multivariate			
	P-value	OR	95% CI for OR		*P*-value	OR	95% CI for OR	
			Lower	Upper			Lower	Upper
SCAI staging at presentation	0.003*	1.671	1.186	2.355				
SO2 ≤93	<0.001*	4.590	2.261	9.319				
GCS ≤13	<0.001*	10.023	3.943	25.480				
SBP ≤80	0.003*	2.965	1.461	6.017				
MAP ≤65	0.001*	3.421	1.653	7.081				
Charlson comorbidity index >3	<0.001*	4.421	2.127	9.191				
CKD	0.001*	4.971	1.896	13.034				
SOFA score >5	<0.001*	21.737	8.858	53.344	<0.001*	8.174	2.656	25.159
APACHE >7	<0.001*	11.687	5.012	27.254	0.008	3.712	1.405	9.803
Hb ≤ 12	0.001*	3.138	1.563	6.302				
Lactate (peak) >3.8	<0.001*	4.177	2.057	8.485				
HCO_3 ≤17.9	<0.001*	5.123	2.357	11.135				
PH ≤7.34	<0.001*	3.755	1.839	7.668				
Creatinine >1.53	<0.001*	8.933	3.496	22.825	0.005*	5.375	1.670	17.299
Baseline GFR ≤47	<0.001*	7.825	3.441	17.794				
Ventilator	<0.001*	8.831	4.044	19.285	0.053	3.083	0.984	9.657
Myocardial blush	0.091	0.700	0.464	1.058				
TIMI flow	0.001*	0.562	0.397	0.796				
OHCA	<0.001*	15.333	3.334	70.522				

*: Statistically significant *P*-value as $P < 0.05$, SCAI: Society for cardiovascular angiography and interventions, SO_2: Oxygen saturation, OR: Odds ratio, GCS: Glasgow coma scale, SBP: Systolic blood pressure, MAP: Mean arterial pressure, CKD: Chronic kidney disease, SOFA: Sequential organ failure assessment, APACHE: Acute physiology and chronic health evaluation, Hb: Hemoglobin, HCO_3: Bicarbonate, PH: Potential of hydrogen (blood pH), GFR: Glomerular filtration rate, TIMI: Thrombolysis in myocardial infarction, OHCA: Out-of-hospital cardiac arrest Table 4 identifies key predictors of in-hospital mortality from univariate and multivariate logistic regression analyses.

Univariate analysis identified significant associations with multiple factors, including low SpO_2, GCS ≤13, SBP ≤80, MAP ≤65, elevated SOFA and APACHE scores, elevated lactate and creatinine, low HCO_3 and pH, CKD, and OHCA (all $P < 0.05$). In multivariate analysis, the strongest independent predictors were SOFA score >5 (OR =8.17, $P < 0.001$), APACHE score >7 (OR =3.71, $P = 0.008$), and creatinine >1.53 (OR =5.38, $P = 0.005$). Ventilator use approached statistical significance ($P = 0.053$)

Table 5: Relation between gender and in hospital and 30 days mortality

		In hospital mortality		Test value	*P*-value	Sig.
		Alive	Died			
		No=107	No=43			
Gender	Female	29 (27.1%)	13 (30.2%)	0.149*	0.699	NS
	Male	78 (72.9%)	30 (69.8%)			
		30 days mortality		Test value	*P*-value	Sig.
		Alive	Died			
		No=94	No=56			
Gender	Female	26 (27.7%)	16 (28.6%)	0.014*	0.904	NS
	Male	68 (72.3%)	40 (71.4%)			

*: ,NS: Not significant, Sig: Significant

Table 6: Relation of single and multivessel intervention to both in hospital and 30-day mortality

Procedure	In hospital mortality	Test value	*P*-value	Sig.		
	Alive	Died				
	No= 107	No= 43				
Single vs multivessel intervention	No	13 (12.1%)	10 (23.3%)	2.915*	0.233	NS
	Single	71 (66.4%)	25 (58.1%)			
	Multi	23 (21.5%)	8 (18.6%)			
Procedure	30 days mortality	Test value	*P*-value	Sig.		
	Alive	Died				
	No= 94	No= 56				
Single vs multivessel intervention	No	12 (12.8%)	11 (19.6%)	2.020*	0.364	NS
	Single	64 (68.1%)	32 (57.1%)			
	Multi	18 (19.1%)	13 (23.2%)			

NS: Not significant, Sig.: Significant

Table 7: Receiver operating characteristics curve to assess SCAI at presentation and at 30 day (table above) and at 24 hours (table below) to detect mortality among the studied patients

Variable	Cut-off point	AUC	Sensitivity	Specificity	+PV	-PV
At presentation	>2	0.631	73.21	46.81	45.1	74.6
At 24 hours	>2	0.843	80.36	84.04	75.0	87.8
Variable	Cut-off point	AUC	Sensitivity	Specificity	+PV	-PV
At presentation	>2	0.637	74.42	44.86	35.2	81.4
At 24 hours	>2	0.889	90.7	80.37	65.0	95.6

AUC: Area under the curve

In our study, the mean patient age was 59.05±12.06 years (range: 26-83 years), closely aligning with the mean age of 58.1±16.2 years reported by Baran et al.[15] Additionally, our cohort was predominantly male (72%), consistent with previous studies highlighting a male predominance in CS populations.

SCAI shock stages were reassessed after 24 hours: 64 patients remained in the same stage, while others shifted significantly. Notably, 29 patients who were initially in stage C improved to stage A, whereas four patients in stage D progressed to stage E. The significant association between changes in SCAI staging and mortality (P = 0.006) reinforces its prognostic value and is consistent with the findings of Baran et al.[15]

Our findings support those of Baran et al.[15] confirming that changes in SCAI staging within the first 24 hours are significantly associated with mortality. This reinforces the 24-hour reassessment as a reliable prognostic tool, emphasizing its critical role in predicting patient outcomes.

In our study, in-hospital mortality was 28.7%, while 30-day mortality reached 37.3%, corresponding to a 62.7% 30-day survival rate. This is higher than the 30% mortality reported by Hanson et al.[16] which may reflect differences in patient characteristics, limited use of mechanical circulatory support, and their exclusion of SCAI stages A and B. Conversely, Ryabov et al.[17] reported a 54% mortality rate, likely influenced by their inclusion of only three patients in SCAI stages A and B. Notably, mortality among patients with SCAI stages C-E in our study was 35%, highlighting the severity of illness in these patients.

In our study, 82% of patients had STEMI; 55.3% presented with anterior STEMI, while 18% had NSTEMI. This contrasts with Pham et al.[18] in which 70.5% of patients had STEMI. Notably, 22 patients initially classified as SCAI stage A in our study deteriorated within 24 hours, whereas Pham et al.[18] reported no stage progression among stage A patients during the same period, highlighting potential differences in patient characteristics and disease progression.

Our study demonstrated that patients with SCAI stage C or worse who improved within the first 24 hours had better short-term and 30-day survival than those initially in stages A or B who deteriorated after 24 hours. This finding aligns with Baran et al.[15] and Morici et al.[19] and underscores the prognostic significance of early hemodynamic improvement in CS.

In our study, 32% of patients required mechanical ventilation, and the in-hospital mortality rate was 79.1%, highlighting a poor prognosis. Similarly, Morici et al.[19] reported a 30.8% ventilation rate in a larger cohort of 237 patients, emphasizing the critical nature of this intervention.

Serum lactate emerged as a strong independent predictor of mortality, especially in patients with advanced SCAI stages. Elevated lactate reflects tissue hypoperfusion and impaired lactate clearance, making it a practical and dynamic tool for risk stratification. Our findings mirror those of Jentzer et al.[20] reinforcing lactate's utility not only as a biomarker of shock severity but also as a guide for therapeutic escalation and response assessment.

Our study demonstrated that OHCA was associated with significantly worse. The outcomes showed a low 30-day survival rate. This aligns with Sarma et al.[21] who emphasize the poor prognosis and high mortality risk among OHCA patients. These findings highlight the critical need for early intervention and advanced supportive measures to improve survival in this high-risk group. In our study, revascularisation was performed only in patients with a good neurological prognosis after OHCA.

Angiographic outcomes also had a strong impact on mortality. Achieving both optimal TIMI flow grade III and myocardial blush grade III was significantly associated with improved survival. This affirms prior studies by Overtchouk et al.[22] and Mehta et al.[23] which demonstrate the importance of timely and effective reperfusion. These findings advocate for a procedural goal beyond simple vessel patency: ensuring full microvascular reperfusion may be critical in improving outcomes.

Multivessel coronary interventions were significantly more frequent in patients classified as SCAI stages D and E, reflecting the increased complexity and severity of their coronary artery disease. This finding underscores the progression of CS and its association with extensive myocardial ischemia requiring aggressive revascularization strategies. However, performing multivessel interventions in critically ill patients presents challenges, as it may increase procedural risk. These observations warrant further investigation into optimizing revascularization approaches for patients with advanced CS.

The lack of availability of mechanical circulatory support in Egypt resulted in the low number of patients in our study who received such support: only 2% (one ECMO and one intra-aortic balloon pump) were placed on mechanical circulatory support (MCS), in contrast to Koester et al.'s[24] report of US trends among patients presenting with acute coronary syndrome complicated by CS, in which 44.4% were placed on MCS devices. The in-hospital mortality rate in their study was 42.9%.[24]

Study Limitations

Despite our findings, several limitations should be considered. The generalizability of the results may be restricted because the study was conducted at a single centre and had a limited sample size. Additionally, the lack of long-term follow-up and the unavailability of mechanical circulatory support devices could have influenced patient outcomes. Furthermore, delayed presentations may have impacted disease severity and treatment efficacy.

CONCLUSIONS

The SCAI shock classification effectively predicts in-hospital and 30-day mortality in patients with AMI and CS. Higher SCAI stages correlate with worse hemodynamics, more severe organ dysfunction, and increased mortality risk. Reassessment at 24 hours enhances prognostic accuracy, identifying patients at risk of deterioration. Early hemodynamic improvement is associated with better outcomes, emphasizing the need for timely intervention.

Ethics

Ethics Committee Approval: The study protocol was approved by the Scientific and Ethical Committee of Ain Shams University Faculty of Medicine (approval number: FMASU MS161/2024, date: 03.03.2024).

Informed Consent: All participants provided informed consent; privacy and confidentiality were ensured.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Y.A., W.E., Concept: Y.A., W.E., Design: Y.A., W.E., M.S.A., Data Collection or Processing: Y.A., M.E.I., M.S.A., Analysis or Interpretation: M.E.I., W.E., M.S.A., Literature Search: M.E.I., W.E., M.S.A., Writing: Y.A., M.E.I., M.S.A.

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RESEARCH ARTICLE

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A Study of Clinical Profile, Chest X-ray, ECG Changes, and 2D Echocardiography in Patients with Chronic Cor Pulmonale

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Abstract

Background and Aim: Chronic obstructive pulmonary disease may result in chronic cor pulmonale, which is defined as right ventricular dilatation and/or hypertrophy resulting from pulmonary hypertension. A non-spesific clinical presentation and limited access to advanced diagnostic tools in resource-constrained settings make early diagnosis challenging. To evaluate the clinical profile and diagnostic findings from chest radiography, electrocardiography (ECG), and two-dimensional echocardiography in patients with clinically confirmed chronic cor pulmonale.

Materials and Methods: A retrospective observational study was conducted at a tertiary care center from October 2016 to September 2018. A total of 50 patients aged 30 to 80 years with clinically diagnosed chronic cor pulmonale were enrolled. Clinical symptoms, radiographic changes, ECG findings, pulmonary function tests (PFTs), and echocardiographic parameters were studied.

Results: The mean age of patients in our study was 51.7 years, with a male predominance (92%). The most common presenting features included breathlessness (100%), productive cough (100%), swelling of the feet (86%), and loss of appetite (92%). Chronic bronchitis with emphysema was the most frequent etiology, accounting for 58% of cases. Chest X-rays revealed chronic bronchitis with emphysema in 58% of patients, increased transverse cardiac diameter in 40% of patients, and a right descending pulmonary artery diameter greater than 16 mm in 62% of patients. ECG findings included right axis deviation (86%), P pulmonale (74%), low-voltage QRS complexes (52%), and arrhythmias (72%). Obstructive patterns were observed in 96% of PFTs. Echocardiography demonstrated dilation of the right ventricle and right atrium in all patients, right ventricular hypertrophy in 84% of patients, pulmonary hypertension in 90% of patients, and tricuspid regurgitation in 90% of patients. Moderate-to-severe pulmonary hypertension was observed in 74% of patients.

Conclusion: In the present study, chronic bronchitis with emphysema was the predominant cause of chronic cor pulmonale among middle-aged males. Multimodal assessment using clinical, radiographic, ECG, and echocardiographic findings enables early diagnosis. Echocardiography serves as a critical tool for evaluating right heart involvement and guiding timely intervention in chronic cor pulmonale.

Keywords: Cor pulmonale, electrocardiography, echocardiography, pulmonary hypertension, right ventricle

INTRODUCTION

Cor pulmonale is characterized by right ventricular hypertrophy (RVH) and/or dilatation resulting from elevated resistance or hypertension in the pulmonary circulation, attributable to conditions that impair lung function and structure.[1,2] The primary etiology of cor pulmonale is chronic obstructive pulmonary disease (COPD), followed by idiopathic pulmonary fibrosis and chronic thromboembolic pulmonary hypertension.[3]

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These disorders typically cause persistent hypoxemia and/or remodeling of the pulmonary circulation, necessitating that the right ventricle (RV) adapt to the increased mechanical load required to pump blood through the lungs.[3] Echocardiography is the primary non-invasive technique for the diagnosis and monitoring of cor pulmonale.[4,5]

However, structural and functional adaptation of the RV in cor pulmonale is often detected late in clinical practice due to its subtle and progressive nature.[6] Right heart dysfunction is often underdiagnosed until the disease reaches an advanced stage because early symptoms, such as exertional dyspnea or fatigue, are commonly mistaken for primary pulmonary disease.[3] Irrespective of left ventricular function or pulmonary parameters, right heart impairment is known to predict adverse outcomes in patients with chronic lung disease.[5,7] Furthermore, cor pulmonale in COPD has been linked to higher hospitalization rates and an increased healthcare burden, particularly in low- and middle-income countries. In addition to echocardiography, chest radiography and electrocardiography (ECG) continue to play an essential role in the initial diagnosis, particularly in resource-limited settings.[7,8] Chest X-rays can reveal features such as cardiomegaly, enlarged pulmonary arteries, and hyperinflated lungs, while ECG may detect right axis deviation (RAD), P pulmonale, and RVH all of which are important indirect indicators of cor pulmonale. A comprehensive understanding of these diagnostic modalities and of their correlations with clinical features is crucial for early recognition, timely intervention, and long-term management of patients with chronic respiratory illnesses. Given the high incidence of COPD and the prognostic importance of RV dysfunction, cor pulmonale is a critical clinical entity associated with significant morbidity and mortality. This study was designed to evaluate the clinical and diagnostic profiles of patients with clinically confirmed chronic cor pulmonale and to assess the characteristic findings on chest radiography, ECG, and two-dimensional (2D) echocardiography.

METHODS

Study Design and Population

This was a retrospective observational study conducted at a tertiary care center between October 2016 and September 2018. Inclusion criteria were adult patients aged 30 to 80 years who presented with clinical features suggestive of chronic cor pulmonale, with the diagnosis subsequently confirmed by chest X-ray, ECG, and 2D transthoracic echocardiography.

Exclusion criteria include patients with a primary diagnosis of bronchial asthma; interstitial lung disease; known left ventricular systolic dysfunction (e.g., ischemic heart disease); poorly controlled hypertension; significant valvular heart disease; congenital heart disease; and poor echogenic windows that precluded echocardiographic examination.

Data Collection and Methodology

Every patient who was enrolled received comprehensive clinical evaluations, including a detailed medical history and a physical examination for signs of right heart failure. Posteroanterior chest X-rays were used to measure transverse heart diameter, right descending pulmonary artery diameter, prominence of the pulmonary conus, and pulmonary artery dilatation. Pulmonary function tests (PFTs) were performed using a computerized spirometer. This was done to detect obstructive lung diseases. Based on PFTs, patients were classified as having restrictive, obstructive, or mixed ventilatory abnormalities. To identify cor pulmonale characteristics such "P" pulmonale, RAD, RVH, and right bundle branch block (RBBB), an ECG was conducted. To evaluate right ventricular dilatation, pulmonary hypertension, and tricuspid regurgitation (TR), 2D echocardiography was performed using a 3.5 MHz transducer, primarily in the parasternal long-axis view. All clinical, radiological, and echocardiographic data were recorded on a standardized pro forma and compiled into a master chart for analysis. RVH is defined as the thickening of the right ventricular wall that develops in response to increased afterload due to pulmonary hypertension, and it may serve as an early indicator of disease. A wall thickness greater than 5 mm is considered abnormal. RA dilation is the enlargement of the right atrium, the heart's upper chamber that receives deoxygenated blood from the body.

Statistical Analysis

SPSS software (version 20, SPSS, Inc., Chicago, IL, USA) was used to analyze the data. The mean and standard deviation were used to characterize continuous variables, whereas frequency counts and percentages were used to characterize categorical variables.

Ethical Statement

This research was authorized by the Institutional Human Ethics Committee of MNR Medical College & Hospital (decision number: ECR/834/1/TG/2016, date: 26.11.2016). All patients provided written informed consent, and the study was approved by the institutional ethics committee.

RESULTS

The study included 50 patients with a diagnosis of chronic cor pulmonale. The mean age of the patients in the study was 51.7 years; 92% were male, indicating a significant male predominance. Nearly half of the patients (48%) had been symptomatic for between two and five years. All patients

presented with cough with expectoration and with exertional breathlessness. Constitutional symptoms were also prevalent, with loss of appetite reported in 92% of patients, swelling of the feet in 86%, and abdominal pain in 82%. Demographic and clinical characteristics (n=50) are presented in Table 1.

On general physical examination, pedal edema, cyanosis, and digital clubbing were observed in 86%, 64%, and 20% of patients, respectively. All patients (100%) had respiratory signs, including tachypnea, reduced chest expansion, rhonchi, and crepitations. Cardiovascular examination findings included a loud second pulmonary heart sound (P2) in 96% of patients, a left parasternal heave in 74%, and raised jugular venous pressure (JVP) in 72%. General physical, respiratory, and cardiovascular examinations (n=50) are presented in Table 2.

Chronic bronchitis with emphysema was the most frequent underlying etiology, identified in 58% of the study population. In 58% of patients, chest radiography showed characteristics suggestive of emphysema and chronic bronchitis. A total of 62% of patients had a right descending pulmonary artery diameter greater than 16 mm, and 40% had cardiomegaly with an enlarged transverse cardiac diameter. ECG findings in patients showed a high prevalence of right heart strain patterns, with P pulmonale present in 74%, RAD in 86%, low-voltage QRS complexes in 52%, and cardiac arrhythmias in 72%. PFTs reported obstructive pulmonary function in 96% of patients. Etiological factors, radiographic and ECG findings, and PFTs in the study population (n=50) are shown in Table 3.

Table 1: Demographic and clinical characteristics (n=50)

Parameters	n (%)
Age distribution	
30-39 years	8 (16%)
40-49 years	11 (22%)
50-59 years	21 (42%)
60-69 years	7 (14%)
70-79 years	3 (6%)
Mean age	51.7 years
Gender	
Male	46 (92%)
Female	4 (8%)
Duration of illness	
≤1 year	1 (2%)
2-5 years	24 (48%)
6-10 years	15 (30%)
11-20 years	8 (16%)
≥21 years	2 (4%)
Presenting symptoms	
Cough with expectoration	50 (100%)
Breathlessness	50 (100%)
Swelling of feet	43 (86%)
Loss of appetite	46 (92%)
Pain abdomen	41 (82%)
Fever	15 (30%)
Hemoptysis	10 (20%)
Palpitation	9 (18%)
Chest pain	5 (10%)

Table 2: General physical, respiratory and cardiovascular examinations (n=50)

Parameters	n (%)
General physical examination	
Cyanosis	32 (64%)
Clubbing	10 (20%)
Pedal edema	43 (86%)
Respiratory signs	
Tachypnoea	50 (100%)
Barrel-shaped chest	36 (72%)
Decreased chest expansion	50 (100%)
Decreased breath sounds	38 (76%)
Rhonchi	50 (100%)
Crepitations	50 (100%)
Cardiovascular signs	
Raised jugular venous pressure	36 (72%)
Left parasternal heave	37 (74%)
Dullness in left 2nd intercostal space	31 (62%)
Loud second pulmonary heart sound (loud P2)	48 (96%)
Tricuspid regurgitation	24 (48%)

All patients had RVH and right atrial (RA) dilatation on echocardiography. Pulmonary hypertension was detected in 90% of the patients; severity was moderate in 32% and severe in 42%. TR was also noted in 90% of patients: 40% had mild TR, 20% had moderate TR, and 30% had severe TR. The right ventricular internal diameter at end-diastole (RVIDED) ranged from 3.4 to 3.8 cm in 50% of patients. Echocardiographic findings (n=50) are presented in Table 4.

DISCUSSION

By offering valuable insights into the clinical presentation and diagnostic features of the syndrome, as observed in a resource-limited tertiary care setting, the current study emphasizes the burden of chronic cor pulmonale among patients with chronic pulmonary disease. In our cohort of 50 patients diagnosed with chronic cor pulmonale, the mean age was 51.7 years, and there was a pronounced male predominance (92%). This aligns with the findings of Goswami et al.[6] who reported a mean age of 54.87±13.76 years and a male predominance of 61%, which highlights the greater vulnerability of males, possibly due

Table 3: Etiological factors, radiographic findings, electrocardiographic findings, and pulmonary function test in the study population (n=50)

Parameters	n (%)
Etiology	
Chronic bronchitis with emphysema	29 (58%)
Bronchial asthma	7 (14%)
Bronchiectasis	5 (10%)
Old pulmonary tuberculosis	7 (14%)
Kyphoscoliosis	2 (4%)
Chest X-ray findings	
Chronic bronchitis with emphysema	29 (58%)
Enlarged transverse cardiac diameter	20 (40%)
Prominent pulmonary conus	17 (34%)
Right descending pulmonary artery >16 mm	31 (62%)
Bronchiectasis	5 (10%)
Old pulmonary tuberculosis	7 (14%)
Kyphoscoliosis	2 (4%)
Electrocardiographic findings	
P pulmonale	37 (74%)
RAD	43 (86%)
RVH	21 (42%)
RBBB	1 (2%)
Low-voltage complexes	26 (52%)
Arrhythmias	36 (72%)
Pulmonary function tests	
Obstructive	48 (96%)
Restrictive	2 (4%)

RAD: Right axis deviation, RVH: Right ventricular hypertrophy, RBBB: Right bundle branch block

Table 4: Echocardiographic findings (n=50)

Parameters	n (%)
Echocardiographic findings	
Right ventricular enlargement	50 (100%)
Dilated right atrium	50 (100%)
Right ventricular hypertrophy	42 (84%)
Right ventricular systolic dysfunction	36 (72%)
Pulmonary hypertension	45 (90%)
Tricuspid regurgitation	45 (90%)
Right ventricular internal diameter in end-diastole	
2.3-2.8 cm	0 (0%)
2.9-3.3 cm	6 (12%)
3.4-3.8 cm	25 (50%)
>3.8 cm	19 (38%)
Pulmonary arterial hypertension	
Mild (30-50 mmHg)	8 (16%)
Moderate (50-70 mmHg)	16 (32%)
Severe (>70 mmHg)	21 (42%)
Severity of tricuspid regurgitation	
Mild	20 (40%)
Moderate	10 (20%)
Severe	15 (30%)
Absent	5 (10%)

to higher smoking rates and occupational exposures in this demographic group. The majority of our patients had a history of chronic illness spanning 2-10 years, reflecting the insidious progression of underlying pulmonary disease before the development of cardiac complications. Clinical manifestations were dominated by productive cough (100%), breathlessness (100%), peripheral edema (86%), and loss of appetite (92%). These are classical features of right-sided heart failure secondary to chronic hypoxic pulmonary disease. A previous study by Divya et al.[9] also noted similar symptoms: 100% of their patients presented with productive cough and pedal edema, though their reported frequency of loss of appetite was lower, at 30%.

Physical signs such as cyanosis (64%) and cardiovascular signs such as elevated JVP (72%) were predominant in our study. A previous study by Divya et al.[9] reported cyanosis in 39% of patients and raised JVP in 75% of patients. A loud pulmonary component of the second heart sound (P2), present in 96% of our cohort, is a well-established indicator of pulmonary hypertension and right ventricular strain, as documented in prior studies.[6,9,10]

In our analysis, chronic bronchitis with emphysema accounted for 58% of cases, with bronchial asthma, old pulmonary tuberculosis, and bronchiectasis followed closely. Goswami et al.[6] reported a slightly higher rate of chronic bronchitis/emphysema (75%) and post-tuberculosis or bronchiectatic disease in approximately 20% of patients. Radiographic results from our study showed that 58% of patients had emphysematous changes, 40% had an increased transverse heart diameter, and 62% had a right descending pulmonary artery larger than 16 mm. These are consistent with chronic pulmonary vascular remodeling and right heart strain. Consistent with our findings, Jatav et al.[10] reported radiological findings, including emphysema in 72% of patients, cardiomegaly in 20% of patients, and a prominent right descending pulmonary artery in 30% of patients, supporting the high diagnostic yield of chest radiographs in cor pulmonale when interpreted alongside clinical findings.

In this study, ECG analysis showed P pulmonale in 74% patients, RAD in 86%, low-voltage complexes in 52%, and arrhythmias in 72% of patients. A similar study by Goswami et al.[6] reported

P pulmonale in 80% of patients, RAD in 81.25% of patients, low-voltage complexes in 27.5% of patients, and arrhythmias in 5% of patients. Compared with Goswami et al.[6] who noted a lower prevalence of arrhythmias (5%), our findings suggest a higher burden of electrical instability, possibly reflecting more advanced disease at presentation. The relatively low-incidence of RVH and RBBB in our cohort may reflect under-detection due to ECG's limited sensitivity for right-sided structural changes. In our patients, 96% displayed an obstructive pattern on spirometry. In line with our findings, Goswami et al.[6] reported an obstructive pattern on spirometry in 92.5% of patients.

In our study, echocardiography revealed RA and RV dilation in 100% of patients, RVH in 84% of patients, and pulmonary hypertension in 90% of patients. In contrast, Jatav et al.[10] observed RA/RV dilation in only 43% of patients and RVH in 42% of patients. Higher detection rates in our study may be due to improved imaging protocols or more advanced disease. The RVIDED exceeded 3.4 cm in 88% of patients, reinforcing the widespread impact of chronic pulmonary pressure overload. Similarly, Goswami et al.[6] reported ≥3.4 cm RVIDED in 73.75% of patients. Most of our patients (74%) had moderate-to-severe pulmonary arterial hypertension. Our finding was consistent with that of Jatav et al.[10] who reported moderate-to-severe pulmonary arterial hypertension in 77.5% of patients, reiterating the central role of pulmonary hypertension in the pathophysiology and clinical course of cor pulmonale.

Overall, our findings corroborate and expand upon existing literature, reinforcing the diagnostic importance of integrating clinical evaluation with radiographic, ECG, and echocardiographic assessments. Early recognition of cor pulmonale in patients with chronic respiratory disease, especially COPD, remains essential, as timely initiation of pulmonary vasodilators, oxygen therapy, and lifestyle modifications can improve functional capacity and outcomes. To improve the prognosis and treatment of patients with chronic cor pulmonale, future research should concentrate on integrating longitudinal follow-up and RV function metrics, including tricuspid annular plane systolic excursion and RV strain.

Study Limitations

This study has certain limitations. Because this was a retrospective, single-center study, its findings may not be generalizable to the broader population. Additionally, the lack of invasive hemodynamic confirmation and the absence of longitudinal follow-up restrict the evaluation of disease progression and therapeutic outcomes. Prospective, multicenter studies with serial echocardiographic assessments and long-term follow-up are warranted.

CONCLUSION

The findings of this study indicate that the most frequent underlying cause of chronic cor pulmonale is chronic bronchitis with emphysema, predominantly affecting males older than 40 years. Common clinical manifestations observed included breathlessness, productive cough, swelling of the feet, and signs of right-sided heart failure. Radiographic and ECG findings, such as emphysematous changes, cardiomegaly, P pulmonale, and RAD, were consistent across cases. Echocardiography reliably demonstrated RA and RV dilatation, pulmonary hypertension, and TR. For individuals with chronic respiratory diseases, our results highlight the value of early, multimodal examination, particularly echocardiography, for prompt diagnosis and treatment of chronic cor pulmonale.

Ethics

Ethics Committee Approval: This research was authorized by the Institutional Human Ethics Committee of MNR Medical College & Hospital (decision number: ECR/834/1/TG/2016, date: 26.11.2016).

Informed Consent: All patients provided written informed consent.

Footnotes

Authorship Contributions

Surgical and Medical Practices: M.Z.U.H., P.K., S.M.H.B. Concept: P.K., S.M.H.B., Design: M.Z.U.H., P.K., Data Collection or Processing: M.Z.U.H., P.K., Analysis or Interpretation: M.Z.U.H., P.K., S.M.H.B., Literature Search: M.Z.U.H., P.K., Writing: M.Z.U.H., P.K.

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LETTER TO THE EDITOR

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Is a New Cardiac Public Health Problem Rising: Jamaica?

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Keywords: Jamaica, bonzai, myocardial infarction, heart failure

To the Editor,

As is the case worldwide, drug use among young people remains an important public health concern in our country. In this letter, we wish to highlight the growing use of Jamaica, a type of synthetic cannabinoid that has recently become widespread among young men due to its relatively low cost. Alarmingly, we have observed serious cardiovascular complications associated with its use.

Case 1: A 28-year-old male patient presented to the emergency department complaining of chest pain. The patient, who had ST elevation in the anterior leads on electrocardiogram (ECG), underwent emergency coronary angiography with a diagnosis of acute anterior myocardial infarction. A 4.5x24 mm drug-eluting stent was directly implanted into a totally thrombosed lesion in the mid left anterior descending. The stent was post-dilated with a 5.0x12 non-compliant balloon. Following a successful primary percutaneous intervention, the patient was monitored in the intensive care unit. During the investigation of the aetiology, it was learned that he had used bonzai in the past year, but used Jamaica regularly for the last 6 months because it was cheaper. The patient, who continued treatment for a cardiac condition, was referred to the psychiatry department for addiction therapy.

Case 2: A 27-year-old male patient presented to the emergency department complaining of nausea, vomiting, and abdominal pain. Blood tests performed in the emergency department showed glucose was 758 mg/dL; urine ketones were negative. He was admitted to hospital with a new diagnosis of diabetes mellitus. During his hospitalisation, the patient had dyspnea and low oxygen saturation. Cardiological examination revealed sinus tachycardia on ECG, and echocardiography was performed, which showed left atrium: 4.1 cm, left ventricular end-diastolic circumferential: 6 cm, ejection fraction (EF) 20%, mitral regurgitation grade 2, and tricuspid regurgitation grade 3. Systolic pulmonary artery pressure was 45 mmHg. The patient's previous records showed that an echocardiogram performed two years earlier had revealed an EF of 65%. The patient was admitted to the intensive care unit with a new diagnosis of heart failure and was started on an angiotensin-converting enzyme inhibitor, sodium-glucose cotransporter 2 inhibitor, mineralocorticoid receptor antagonist, diuretic, ivabradine, and beta-blocker, after hypervolaemia resolved. Etiological investigation, in the young patient revealed that he had been using Jamaica regularly for the past 7 months. The patient was referred to the psychiatry department for addiction therapy.

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In the literature, we have not found any studies that attracted our attention regarding the cardiovascular side effects associated with acute or chronic use of the substance sold under the street name "Jamaica". We have observed that it is becoming increasingly prevalent in our country and that may contain many synthetic cannabinoid groups such as JWH, AM, and AB. The literature reports cardiovascular effects associated with the acute toxicity of other synthetic cannabinoids such as bonzai, spice, and K2, including acute myocardial infarction, atrial fibrillation, and ventricular arrhythmia cases associated with the acute toxicity of other synthetic cannabinoids such as bonzai, Spice, and K2 and our findings of no other cause in the aetiology of these two patients lead us to consider acute myocardial infarction and dilated cardiomyopathy associated with Jamaica in these two cases.[1-5]

In conclusion, unexplained arrhythmias, myocardial infarction, or new-onset heart failure in young patients should prompt physicians to inquire not only about well-known synthetic cannabinoids but also other locally available variants such as Jamaica. Public health initiatives aimed at curbing synthetic cannabinoid use are urgently needed.

Footnotes

Authorship Contributions

Data Collection or Processing: E.P., E.E., B.D., H.K., Literature Search: E.P., H.K., Writing: E.P., E.E.

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EDITORIAL COMMENT

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HbA1c and Coronary Artery Disease Severity: Insights from SYNTAX Score II in Diabetic Patients

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Keywords: HbA1c, Coronary artery disease severity, SYNTAX score II

Ischemic heart disease, the leading global cause of death with 9.1 million fatalities in 2021, disproportionately affects patients with diabetes, amplifying the need to identify modifiable risk factors.[1] The study by Viruthagiri et al.[2] published in this issue of the International Journal of the Cardiovascular Academy, provides robust evidence linking glycemic control to the anatomical severity of coronary artery disease (CAD) in a diabetic cohort. The authors conducted a prospective observational study of 121 diabetic patients with angiographically confirmed CAD at SRM Medical College Hospital in India. The study utilized the SYNTAX score II (SSII)—which stratifies disease complexity into low (<22), intermediate (23-32), and high (≥33) risk categories—to determine the relationship between metabolic parameters and coronary complexity.[2]

The Aggressive Link: Glycated Hemoglobin (HbA1c) and High-risk CAD

The findings confirm that poor metabolic control correlates directly with advanced coronary disease. Patients exhibited a high mean HbA1c level (8.53±1.68%), which significantly predicted severe CAD ($P = 0.040$). Crucially, each 1% increase in HbA1c raised the odds of belonging to the high-risk SSII group by 62.9% [odds ratio (OR) =1.62, $P = 0.014$].

This relationship is not isolated; the analysis underscores a pervasive cardiorenal-metabolic interplay. Longer diabetes duration (OR =1.13, $P = 0.049$); reduced left ventricular ejection fraction (LVEF) (OR =0.0004, $P = 0.019$); and declining creatinine clearance (OR =0.96, $P = 0.001$) were also identified as independent predictors of worsening SSII (Table 1). These results strongly align with global evidence linking chronic hyperglycemia to accelerated atherosclerosis via endothelial dysfunction and inflammation.[3]

Therapeutic Mandate

The utility of the SSII in risk stratification supports its vital role in guiding revascularization decisions (percutaneous coronary intervention vs. coronary bypass grafting). The study compellingly highlights HbA1c as a critical therapeutic target; achieving and maintaining tight glycemic control is essential to mitigate the progression of complex coronary anatomy. Furthermore, the strong associations involving LVEF and creatinine clearance support integrated cardiorenal management to address the systemic nature of the disease in patients with diabetes.

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***Table 1: Association between key risk factors and severe coronary disease (SYNTAX score II ≥33)**

Risk comparison	Independent risk factor	Odds ratio	*P*-value	Clinical implication
Low-risk vs. high-risk	1% increase in HbA1c	1.63	0.014	Poor glycemic control increases the odds of having the most severe CAD by approximately 63%
Low-risk vs. high-risk	1 year increase in diabetes duration	1.13	0.049	Each additional year of disease increases the risk of severe CAD by 13%
Low-risk vs. high-risk	Decreased LVEF	0.0004	0.019	Reduced cardiac function is a strong independent predictor of severe CAD
Low-risk vs. high-risk	Declining creatinine clearance	0.96	0.001	Declining renal function significantly increases the likelihood of complex CAD

*: Summary of the multinomial logistic regression analysis highlighting the independent predictors for high-risk coronary artery disease (SYNTAX score II ≥33) compared to the low-risk group, HbA1c: Glycated hemoglobin, LVEF: Left ventricular ejection fraction, CAD: Coronary artery disease

While limitations such as the single-center design and the exclusion of well-controlled diabetics restrict the generalizability of the findings, the conclusions offer valuable insights for clinicians. This study underscores the need for multicenter trials to refine personalized strategies for patients with diabetes, ultimately aiming to reduce the global burden of advanced CAD.

Ethics

Financial Disclosure: The author declared that this study received no financial support.

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CASE REPORT

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Lev's Disease Presenting with Complete Atrioventricular Block in a Patient with Severe Aortic Stenosis: A Case Report

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Abstract

Lev's disease is an age-related degenerative conduction disorder characterized by progressive fibrosis and calcification of the His-Purkinje system and adjacent structures. It typically manifests in elderly patients as advanced atrioventricular (AV) block and is often associated with calcific valvular disease. This case report describes a 74-year-old man with a history of stable, non-obstructive coronary atherosclerosis who presented with dizziness and was found to have complete AV block with a ventricular escape rhythm of 36 beats per minute. Transthoracic echocardiography demonstrated severe aortic stenosis with an aortic valve area of 0.96 cm². Contrast-enhanced computed tomography revealed extensive aortic valve calcification extending into the interventricular septum, raising strong clinical-radiological suspicion of Lev's disease due to the anatomical proximity of the His bundle. Surgical risk stratification using Society of Thoracic Surgeons and European System for cardiac operative risk evaluation II indicated an elevated operative risk. Given the patient's advanced age, severe symptomatic aortic stenosis, and persistent conduction abnormality, the multidisciplinary Heart Team recommended transcatheter aortic valve implantation (TAVI) rather than surgical replacement. The patient underwent successful TAVI followed by dual-chamber pacemaker implantation for persistent AV block and remained pacemaker-dependent with a stable rhythm and resolution of symptoms during follow-up. This case underscores that degenerative conduction disease may remain clinically silent until irreversible AV block occurs. Unlike most reports describing new-onset conduction disturbances after TAVI, our patient already presented with complete AV block and imaging demonstrated extension of septal calcification involving the conduction system. Although histopathological confirmation and prior serial electrocardiogram were unavailable, this case highlights the potential role of advanced imaging in identifying patients at increased risk for permanent pacemaker dependence, thereby enabling more accurate risk stratification and guiding closer follow-up.

Keywords: Advanced imaging systems, aortic stenosis, atrioventricular block, cardiac computed tomography, lev's disease, transcatheter aortic valve implantation

INTRODUCTION

Severe calcific aortic stenosis (AS) is the most common valvular heart disease in the elderly population; its prevalence increases markedly with age, and it is associated with high morbidity and mortality if untreated.[1] Classical symptoms include dyspnea, angina, and syncope; however, conduction abnormalities are also frequently observed due to the extension of calcification into the interventricular septum and His-Purkinje system. Advanced atrioventricular (AV) block is one of the most serious complications and often requires permanent pacemaker implantation.[2,3]

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Degenerative conduction system disease, historically described as Lenegre's and Lev's diseases, is characterized by progressive idiopathic fibrosis and calcification of the His bundle and bundle branches and typically presents with AV block in elderly patients. In the context of severe AS, the coexistence of valvular calcification and degenerative conduction system disease may accelerate the progression to complete heart block, creating both diagnostic and therapeutic challenges.[4-6]

We present an elderly patient with severe calcific AS who developed advanced AV block attributed to Lev's disease. This case highlights the clinical interplay between degenerative valvular and conduction system pathology and underscores the importance of early recognition and management in high-risk older adults.

CASE REPORT

A 74-year-old male with a history of stable, non-obstructive coronary atherosclerosis (no prior myocardial infarction, percutaneous coronary intervention, or coronary artery bypass surgery) presented to the emergency department with dizziness. A 12-lead electrocardiogram (ECG) revealed complete AV block with a ventricular escape rhythm at 36 beats per minute (Figure 1). Transthoracic echocardiography demonstrated a preserved left ventricular ejection fraction (60%), mild mitral and tricuspid regurgitation, and severe AS with an aortic valve area of 0.96 cm² (Figure 2). The coexistence of high-grade AV block and severe calcific AS raised clinical suspicion for Lev's disease.

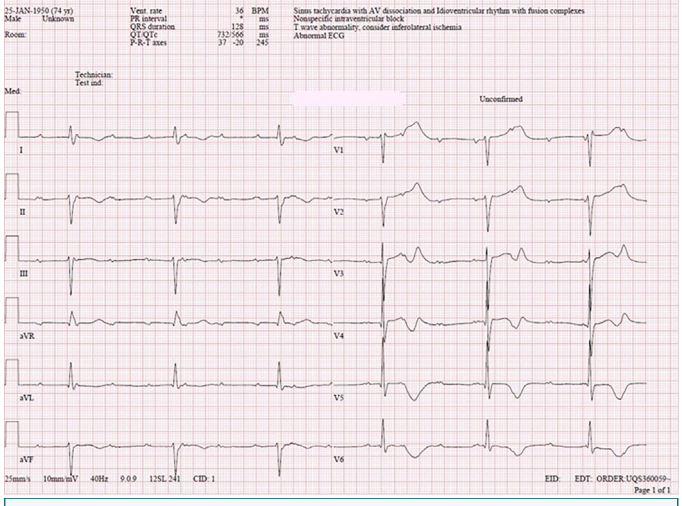

Figure 1. Admission electrocardiogram showing complete atrioventricular (AV) block with a ventricular escape rhythm at 36 beats per minute. There is AV dissociation, with P-waves occurring independently of the QRS complexes. The QRS duration is prolonged (128 ms), consistent with non-spesific intraventricular conduction delay. T-wave abnormalities in the inferolateral leads suggest possible ischemic changes

Contrast-enhanced thoracic computed tomography (CT) revealed extensive calcification of the aortic valve extending toward adjacent cardiac structures, including the interventricular septum (Figure 3). Given the anatomical proximity of the His bundle to the membranous septum, conduction system involvement was strongly suspected. Since histopathological confirmation was not feasible, the diagnosis of Lev's disease was considered on a clinical-radiological basis.

Surgical risk stratification indicated an STS-predicted operative mortality of approximately 4%, a combined morbidity and mortality risk of approximately 13%, and a European System for Cardiac Operative Risk Evaluation II of 5.28%. Considering the patient's advanced age, severe symptomatic AS, and persistent high-grade conduction abnormality, the multidisciplinary Heart Team determined that surgical valve replacement carried excessive risk and recommended transcatheter aortic valve implantation (TAVI) as the preferred strategy.

With temporary transvenous pacing support, a CoreValve/Evolut prosthesis was successfully implanted. Final angiography showed no paravalvular leak, and the procedure was completed without complications. Despite hemodynamic improvement following valve replacement, the complete AV block persisted. A permanent dual-chamber pacemaker was subsequently implanted.

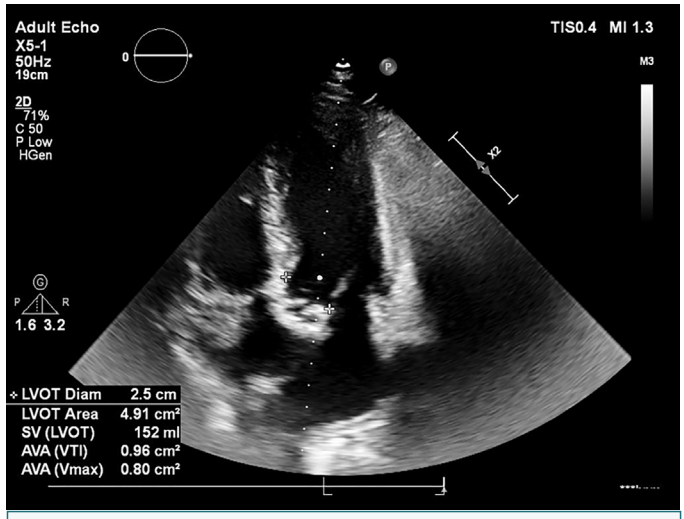

Figure 2. Transthoracic echocardiographic image demonstrating severe calcified aortic stenosis. The LVOT diameter was measured at 2.5 cm, resulting in a calculated area of 4.91 cm² and a stroke volume of 152 mL. Although this value appears relatively high for severe AS, it may reflect preserved systolic function and should be interpreted in clinical context. The aortic valve area was calculated as 0.96 cm² (VTI method) and 0.80 cm² (Vmax method), confirming severe stenosis

LVOT: Left ventricular outflow tract, VTI: Velocity time integral, AS: Aortic stenosis

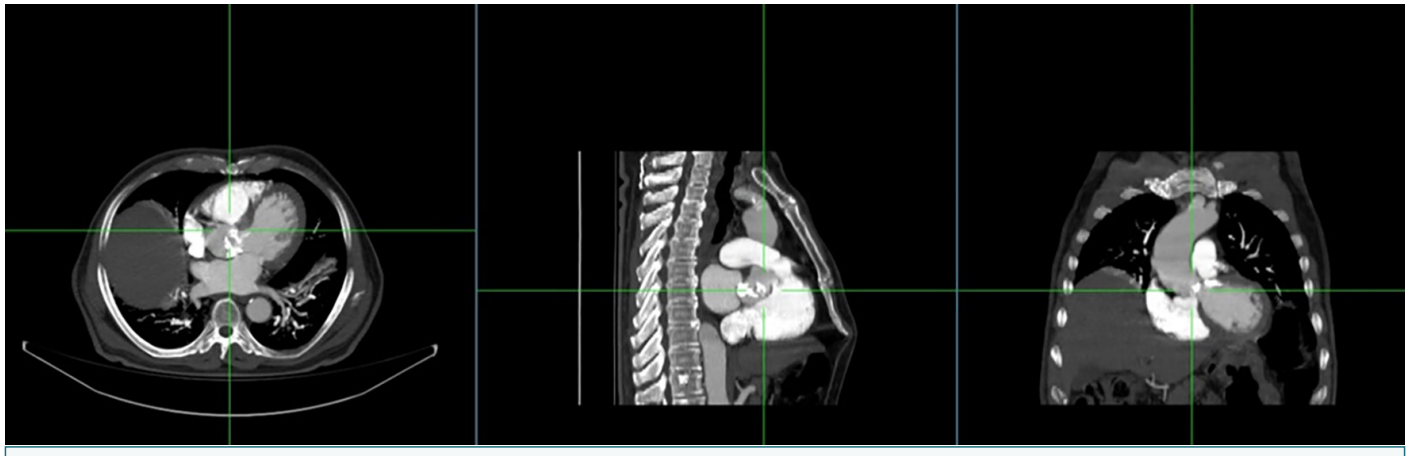

Figure 3. Contrast-enhanced thoracic computed tomography demonstrating severe calcification of the aortic valve with extension toward the interventricular septum. Multiplanar reconstructions (axial, sagittal, and coronal views) show calcific infiltration from the aortic root into adjacent cardiac structures, raising the possibility of conduction system involvement due to proximity to the His bundle. These findings support the suspicion of a degenerative conduction disorder coexisting with severe aortic stenosis

The patient's recovery was uneventful, with complete resolution of symptoms and maintenance of stable cardiac rhythm. At 1- and 3-month follow-up visits, the patient remained pacemaker-dependent, clinically stable, and free of recurrent symptoms.

Written informed consent was obtained from the patient for the publication of this case report and the accompanying images.

DISCUSSION

This case highlights the intricate relationship between severe AS and advanced AV conduction disturbances, raising a clinical-radiological suspicion of Lev's disease. Lev's disease is characterized by acquired fibrocalcific degeneration of the His-Purkinje system in elderly patients, and should be distinguished from Lenegre's disease, which typically affects younger individuals and is a primary fibrotic process without calcification.[5,6] Unlike ischemic conduction disease, in which scarring is usually confined to areas of prior infarction, Lev's disease involves diffuse calcific infiltration extending into the interventricular septum, as demonstrated by the CT findings in our patient. Although histopathological confirmation would provide definitive proof, it was not pursued in our case due to the limited feasibility of obtaining conduction system tissue, the increased risk of periprocedural complications, and the lack of additional therapeutic benefit, as avoiding potential harm was prioritized. In clinical practice, the diagnosis relies on the integration of clinical features with advanced imaging findings; in this case, these findings were suggestive of Lev's disease.

A unique aspect of this case is that the patient presented with complete AV block prior to intervention. Most reports of conduction disturbances in severe AS emphasize new-onset AV block following TAVI. Our findings suggest that in some cases, the substrate for conduction failure preprocedurally, and that advanced imaging can non-invasively demonstrate septal calcification that directly involves the conduction axis. This highlights the value of CT in risk stratification, complementing echocardiography by not only characterizing valvular pathology but also anticipating electrical complications. Verhemel et al.[7] recently demonstrated that CT-derived parameters such as septal calcification burden and membranous septum length predict permanent pacemaker implantation after TAVI. Similarly, Pagnesi et al.[3] reported that patients requiring pacemakers after TAVI had higher mortality and rehospitalization rates, underscoring the prognostic importance of conduction system assessment.

Electrocardiographic surveillance is another pragmatic tool for detecting progressive conduction disease.[8] Serial ECGs may document gradual PR prolongation or bundle branch involvement, which can signal advancing conduction system degeneration.[9,10] Unfortunately, our patient lacked longitudinal ECG documentation a limitation that reduces diagnostic certainty and has been explicitly acknowledged. In such scenarios, electrophysiological studies could theoretically quantify conduction reserve and confirm diffuse His-Purkinje involvement; however, invasive assessment was not pursued because of the patient's frailty and the urgent need for valve intervention.

From a therapeutic standpoint, management must address both mechanical obstruction and electrical instability. TAVI is increasingly favored in elderly high-risk patients with severe symptomatic AS, but it does not reverse degeneration of the intrinsic conduction system.[11] Indeed, conduction disease often persists or worsens after valve implantation, necessitating permanent pacemaker therapy. Recent registry data indicate that 8-25% of patients require new pacemaker implantation

post-TAVI, particularly those with pre-existing conduction abnormalities or septal calcification.[3,12,13] Importantly, long-term studies show that pacemaker dependence after TAVI is associated with higher mortality and adverse outcomes, reinforcing the clinical relevance of early identification.[13]

CONCLUSION

This case underscores that degenerative conduction disease, such as Lev's disease, may remain clinically silent until irreversible AV block occurs. Unlike most reports describing new-onset conduction disturbances after TAVI, our patient had already presented with complete AV block, and CT demonstrated septal extension of valve calcification, strongly suggesting direct involvement of the conduction system. This finding indicates that advanced imaging before TAVI could help identify patients at higher risk of pacemaker dependence, allowing more accurate risk stratification and closer follow-up.

Ethics

Informed Consent: Written informed consent was obtained from the patient for the publication of this case report and the accompanying images.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Ö.F.Y., C.K., Ö.K.M., H.S., U.A., Concept: Ö.F.Y., C.K., Ö.K.M., H.S., U.A., Design: Ö.F.Y., C.K., Ö.K.M., H.S., U.A., Data Collection or Processing: Ö.F.Y., C.K., Ö.K.M., H.S., Literature Search: Ö.F.Y., C.K., Ö.K.M., H.S., Writing: Ö.F.Y., C.K.

Conflict of Interest: No conflict of interest was declared by the authors.

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