ORIGINAL ARTICLE

CLINICAL PRESENTATION OF CARDIOVASCULAR DISEASES AMONG YOUNG ADULTS IN PESHAWAR, PAKISTAN: A MULTICENTERE CROSS-SECTIONAL STUDY

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Background. Cardiovascular diseases (CVDs) remain the leading cause of morbidity and mortality, and their burden continues to rise among young adults globally. Major risk factors, including hypertension, diabetes, smoking, and physical inactivity, are driving the age of CVDs onset to shift earlier. However, data on the clinical presentation and risk characteristics of CVDs in this population remain limited, especially in resource-limited regions. This study, therefore, describes the clinical presentation of CVDs among young adults. **Methods.** This study was conducted in Peshawar, Pakistan, as a multi-center retrospective cross-sectional study in four public sector tertiary hospitals. Data was extracted from the Hospital Management Information System, using a census sampling technique in accordance with pre-set inclusion criteria, and the analysis was conducted using advanced statistical tests. **Results.** A total of 8,650 records were screened, and 2,090 met the eligibility criteria of young adults and Pakistani nationality. Participants were predominantly male (65%) with a median age of 38 years (IQR 34–42). Hypertension was the most common risk factor (50.5%), followed by diabetes mellitus (38.7%). In young age the 70% of patients were severe Killip classes (III–IV) and the unstable angina was (58.5%). Multivessel coronary artery disease (55%) and high lesion complexity (40% Type C) were frequent. Mean blood pressure was elevated (139.2/82.3 mmHg) and the median hospital stay was 6 days (IQR 4–8). Diabetes mellitus (IRR 1.20; 95% CI 1.09–1.32), hypertension (IRR 1.15; 95% CI 1.05–1.26), and elevated heart rate (IRR 1.08; 95% CI 1.03–1.13) were all associated with extended hospitalisation in a multivariable negative binomial regression. Adherence to protocol-recommended therapy at discharge was over 94%. **Conclusion.** CVDs were associated with modifiable risk factors, and they were often associated with severe clinical and anatomic features. Early detection and treatment may help reduce the disease burden and improve outcomes. Prevention, awareness raising, and early screening are likely needed to curb the growing burden of CVDs in this population.

Keywords: Cardiovascular diseases; Hypertension; Diabetes mellitus; Risk factors; Killip classification; Hospitalisation; Clinical outcomes; Cross-sectional study; Cardiometabolic disorders

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INTRODUCTION

Cardiovascular diseases (CVDs) remain the leading cause of death and disability worldwide. In 2022, they were responsible for an estimated 19.8 million deaths, around 32% of all deaths globally.[1] Most of this mortality – roughly 85% – stems from heart attacks and strokes, and more than three-quarters of these deaths occur in low- and middle-income countries (LMICs).[2] While the majority of deaths from cardiovascular disease occur in older age groups[3], it's important to note that over the past decade, a significant epidemiological change has been seen with CVDs affecting younger individuals[4]. An increasing variety of cardiovascular disease entities are seen in the adult population between ages 18 and 45, including coronary artery disease (CAD), acute myocardial infarction (AMI), hypertension, cardiomyopathies, and arrhythmias.[5] Presentation of these disease entities may be different than what is typically seen in older adults. Younger patients can experience atypical symptoms with acute coronary syndromes, such as epigastric distress, fatigue, or mild chest pain instead of classic severe chest pain with radiation.[6,7]

Much of the increased burden is attributable to the escalating prevalence of modifiable risk factors.[8] Increased urbanisation, globalisation and technology have transformed day-to-day activities, minimising physical activity and leading to more sedentary

behaviours and consumption of unhealthy diets rich in processed foods and sugar-sweetened drinks.[9] These behaviours contribute to obesity, dyslipidaemia, insulin resistance, and hypertension, which each increase cardiovascular risk. Metabolic disease in particular exerts a powerful influence on the risk profile of young adults[10]: of particular concern is the earlier onset of type 2 diabetes mellitus (DM) and metabolic syndrome, both of which hasten atherosclerosis and heighten risk for cardiovascular events[11,12]. Hypertension, which has been dubbed the silent killer, is also being increasingly recognised among young populations and is frequently undetected or poorly controlled due to low awareness, insufficient screening, and poor treatment adherence.[13,14] In addition, genetic predisposition, high prevalence of consanguineous marriage and positive family history add to cardiovascular risk.[15] Inherited disorders discussed in the literature such as familial hypercholesterolemia are highly prevalent and associated with massively increased risk of early events.[16,17] As young adults represent a literate portion of population, the emergence of chronic disease at this time is associated with loss of productive life years, reduction in quality of life and increased health care costs.[18,19] The cost of extended treatment, hospitalisation and rehabilitation burdens patients, families and health systems even more in LMICs.[20]

While the burden of CVDs is progressively accumulating, data on clinical patterns and risk profiles of CVDs among young adults are limited, particularly in resource-limited settings. More knowledge about these patterns would allow for early diagnosis, risk stratification and targeted prevention and management strategies. Hence, this study aimed to identify the clinical patterns and risk profiles of cardiovascular diseases in Peshawar-Pakistan among young adults. These findings can inform existing evidence, local public health policy, and contribute to improve cardiovascular outcomes and reduce overall disease burden in this high risk group.

MATERIAL AND METHODS

This multicenter, retrospective cross-sectional study was based on hospital records from regular patients in four major public-sector tertiary-care hospitals in Peshawar, Pakistan (Hayatabad Medical Complex), Khyber Teaching Hospital, Lady Reading Hospital, and Peshawar Institute of Cardiology). These hospitals are the main cardiovascular disease referral hospitals in Peshawar, Khyber Pakhtunkhwa, Pakistan.

All the records of the eligible patients admitted between September 2021 and August 2024 for each hospital were obtained from the Hospital Management Information System (HMIS). The clinical records covered admissions during the 3-year study period; data extraction, cleaning, and validation took place over a period of 12 months. Data analysis was based on the clinical records. Diagnoses were those recorded by the attending cardiologists at the time of admission and discharge; any supporting information (electrocardiography, cardiac biomarkers, and coronary angiogram reports) was used to support the diagnosis.

The protocol was reviewed and approved by the Ethical Review Board of Khyber Medical University, Peshawar (approval number: KMU/IPHSS/Ethics/2023/EA/0160;21/09/2023). Hospital administrators were given permission to access patients' records. The trial used de-identified retrospective data, and informed consent was not required. All procedures were conducted in accordance with the Declaration of Helsinki.

Using a census sampling approach, every patient who met our eligibility criteria during the study period (18–45 years), who had been admitted with a cardiovascular diagnosis, and had complete records were included. 18–45 years age range is widely used as the age group for “young adults” in cardiovascular epidemiology and in regional studies of premature CAD.[4,5] Although definitions of “young adults” differ among cardiovascular researchers, under 18 are the ages at which traditional risk prediction strategies are most effective, and after which death may cause the greatest social and economic loss. Patients without outcome data, duplicate records, or congenital heart disease were excluded. 2,090 of 8,650 records that were screened, 2,090 patients met the eligibility criteria and were included in the final analysis.

Demographics (age, sex, area of residence), cardiovascular risk factors (hypertension, diabetes mellitus, smoking), admission vital signs, baseline co-morbidities, presentation, coronary angiographic and surgical characteristics, and discharge medications were all extracted. All information was cleaned, coded, and checked for completeness and consistency before analysis.

Continuous variables were examined for normality by visual inspection of histograms and the Shapiro–Wilk test. Means SD and medians are presented with interquartile ranges (IQR). Categorical variables are presented as counts and percentages, respectively, with a 95% Wilson confidence interval (CI). All inferential tests were two-sided and a significance level of = 0.05 was established. Differences between groups in continuous variables (for example, heart rate by sexes) were assessed using Welch’s t-test, which assumes equal variances, where the difference is reported as Cohen’s d. Systolic blood pressure was assessed across the five districts using one-way ANOVA, with 2 as the effect size. Because length of stay in hospitals is a right-skewed count outcome (in days), an ordinary least squares regression was unsuitable and we chose to use a negative binomial

generalised linear model with a log link and reported effects as incidence rate ratios (IRRs) with 95% CIs. The model included age and admission heart rate (both standardised), diabetes mellitus, hypertension, sex, and Killip class as covariates. Model fit was assessed using the Akaike Information Criterion (AIC) and residual deviance. All analyses were conducted using SPSS version 29.

RESULTS

All results below are derived from the single analytic cohort of 2,090 young adults (18–45 years) admitted with cardiovascular conditions. Where an analysis applies to a subgroup, the relevant denominator is stated in the text and in the table footnotes (for example, the angiography cohort, n = 1,932; the PCI cohort, n = 1,371).

A total of 2,090 participants were included in the final analysis (Table 1). Most were male (65%, n = 1,359), and 35% (n = 731) were female. The median age was 38 years (IQR 34–42), confirming a predominantly young cohort. Patients were referred from several districts, most commonly Peshawar, followed by Mardan, Charsadda, and Lower Dir. Hypertension was the most frequent risk factor (50.5%), followed by diabetes mellitus (38.7%) and a current or past smoking history (28.5%). Mean systolic and diastolic blood pressures were 139.2±24.7 mmHg and 82.3±15.3 mmHg, respectively, and the average length of stay was 6.0±2.8 days.

Baseline comorbidities were generally uncommon (Table-1). The most frequent was prior myocardial infarction (9.0%, n =188). All other conditions – including cardiac arrest (0.4%), atrial fibrillation or flutter (0.4%), chronic lung disease (0.4%), cerebrovascular disease (0.3%), chronic liver disease (0.2%), and dialysis dependence (0.1%) – were rare, as were prior heart failure, peripheral arterial disease, and previous valve procedures. Overall, the cohort carried a low burden of established cardiovascular comorbidity.

Figure 1 plots the distribution of Killip classes for patients in each participating centre. Of all Killip classes, 41.4% of patients were Killip Class IV, followed by Class III (28.9%) and Class II (18.8%); the lowest incidence was in Class I (10.8%). Most (70%) of patients (70%) were in the high Killip classes (III and IV), indicating an increased risk of severe cardiac dysfunction at presentation. This was partly due to the high prevalence of tertiary referrals from participating centres. Error bars in Figure 1 are 95% Wilson confidence intervals (confidence intervals).

Table 2 lists the clinical presentation of coronary artery disease. Unstable angina was the most common presentation, with 1,222 (58.5%) patients having unstable angina. The next most common presentation was ST-elevation myocardial infarction within 7 days in 373 (17.8%), and stable angina in 244 (11.7%). Non-ST-elevation myocardial infarction (NSTEMI) was less frequent, with 98 cases (4.7%) within 7 days and 72 cases (3.4%) within 8 to 30 days; only a small number of patients were classified as having no symptoms or angina (0.7%) or symptoms unlikely to be ischaemic (1.1%).

Assessment of chest-pain symptoms identified four categories: typical angina, atypical angina, non-anginal chest pain, and asymptomatic presentations. Typical angina predominated strongly, occurring in 91.2% of the cohort. Atypical angina was seen in 6.7% of cases. Non anginal chest pain (1.3%) and asymptomatic presentations (0,8%) were rare. This distribution is typical of the high concentration of symptomatic coronary-related variables in the sampled population and provides a clinical baseline to support the subsequent assessments.

Angiographic evaluation revealed a high prevalence of multivessel disease (Table 3). Double-vessel disease was the most frequent finding (30.7%, n = 593), followed closely by single-vessel disease (29.6%, n = 572). Triple-vessel disease was identified in 24.6% (n = 476), and 15.1% (n = 291) of patients had normal coronary vessels. Most percutaneous coronary interventions (PCIs) were performed electively (74.5%, n = 1,021); emergency and urgent procedures accounted for 17.2% (n = 236) and 8.3% (n = 114), respectively. Staged PCI was the most common indication (51.5%, n = 706), followed by PCI for high-risk NSTEMI or unstable angina (25.2%, n = 346). STEMI-related indications included PCI for unstable STEMI more than 12 hours from onset (9.1%, n = 125), PCI for stable STEMI more than 12 hours from onset (6.1%, n = 84), and primary PCI for STEMI (4.3%, n = 59). Less frequent indications included PCI in stable patients after full-dose thrombolysis (1.3%) and rescue PCI after failed thrombolysis (0.9%).

Lesion morphology (complexity and length) Lesion dimensions showed a wide range of plaque distribution (Figure 2a). Among the subset of patients with available measurements (n = 1,371), the mean lesion length was 20.9 mm. According to the ACC/AHA classification, 40.1% of lesions (n = 550) were Type C, indicating a high degree of procedural complexity, while the remaining 59.9% (n = 821) were Type A or B (non-Type C) (Figure 2b).

Discharge medication use is shown in Table 4. Patients were prescribed aspirin (95.1%, n = 1,987) and clopidogrel (94.6%, n = 1,977) immediately following discharge, consistent with the guidelines. Most patients were prescribed an ACE inhibitor (80.3%, n = 1,678). Non-statin lipid therapy was only used to very minimal effect (0.1%).

Mean heart rate was significantly higher in women than men (86.21 14.15 vs 82.16 14.36 bpm; difference of 4.05 bpm (95% CI 2.77–5.33; t = 6.21, p 0.001) and had a small effect size (Cohen's d = 0.28).

Mean systolic blood pressure ranged between Charsadda (137.2 24.5 mmHg) and the other group (140.6 22.6 mmHg), with the largest number of patients in Peshawar (139.3 24.8 mmHg, n = 868). ANOVA results revealed no difference between districts (F (4, 2085) = 0.92, p = 0.45), and district of residence did not significantly contribute to variance (2 = 0.002).

In the negative binomial model, three covariates independently predicted longer hospitalisation. Diabetes mellitus was associated with a 20% longer expected stay (IRR 1.20, 95% CI 1.09–1.32, $p < 0.001$) and hypertension with a 15% longer stay (IRR 1.15, 95% CI 1.05–1.26, $p = 0.004$). Each one-SD increase in admission heart rate corresponded to an 8% longer stay (IRR 1.08, 95% CI 1.03–1.13, $p = 0.001$). Age (IRR 1.03 per SD, $p = 0.25$), male sex (IRR 1.02, $p = 0.70$), and Killip class (Class III IRR 0.95, $p = 0.44$; Class IV IRR 1.05, $p = 0.43$) were not significant after adjustment. Full results are presented in Table 5.

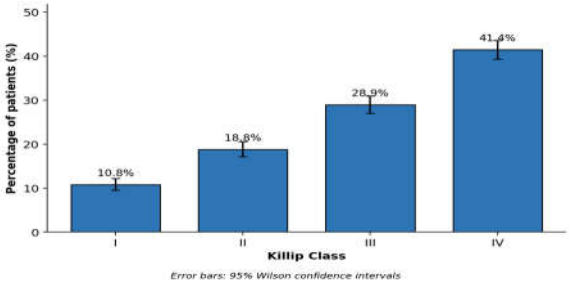
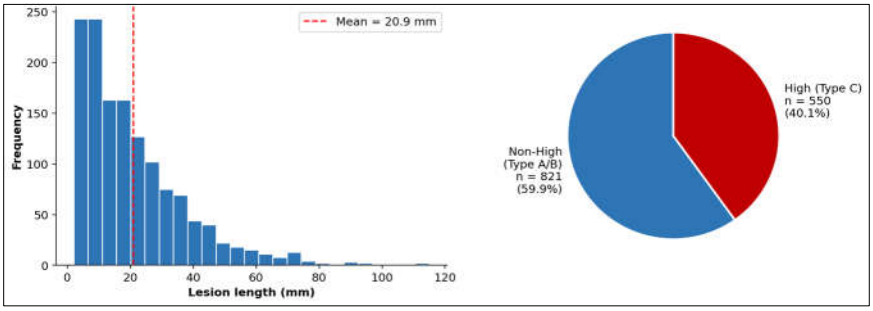


Figure-1: Killip class distribution among the analytic cohort (N = 2,090)



2a. Lesion length distribution (n = 1371) **2b. ACC/AHA lesion complexity (n = 1371)**

Figure-2 Lesion morphology (Lesion length distribution and lesion complexity)

Table-1: Demographics, clinical profile, and baseline comorbidities (N = 2,090)

Characteristic	n (%) / mean ± SD	95% CI / Median (IQR)
Demographics		
Sex – Female	731 (35.0%)	33.0–37.0
Sex – Male	1359 (65.0%)	63.0–67.0
Age (years)	37.7 ± 5.1	38 [34–42]
District – Charsadda	320 (15.3%)	13.8–16.9
District – Lower Dir	225 (10.8%)	9.5–12.2
District – Mardan	390 (18.7%)	17.0–20.4
District – Other	287 (13.7%)	12.3–15.3
District – Peshawar	868 (41.5%)	39.4–43.7
Clinical profile		
Hypertension	1056 (50.5%)	48.4–52.7
Diabetes mellitus	809 (38.7%)	36.6–40.8
Current/ever smoker	595 (28.5%)	26.6–30.4
Heart rate (bpm)	83.6 ± 14.4	84 [74–94]
Systolic BP (mmHg)	139.2 ± 24.7	139 [123–155]
Diastolic BP (mmHg)	82.3 ± 15.3	82 [72–93]
Length of stay (days)	6.0 ± 2.8	6 [4–8]
Baseline comorbidities		
Prior myocardial infarction	188 (9.0%)	7.8–10.3
Cardiac arrest	8 (0.4%)	0.2–0.8
Atrial fibrillation/flutter	9 (0.4%)	0.2–0.8
Chronic lung disease	8 (0.4%)	0.2–0.8
Chronic liver disease	5 (0.2%)	0.1–0.6
Cerebrovascular disease	6 (0.3%)	0.1–0.6
Peripheral arterial disease	1 (0.0%)	0.0–0.3
Prior valve surgery/procedure	2 (0.1%)	0.0–0.3
Currently on dialysis	3 (0.1%)	0.0–0.4

BP, blood pressure; CI, confidence interval; IQR, interquartile range; SD, standard deviation. Proportions are reported with 95% Wilson confidence intervals.

Table-2: Coronary artery disease presentation and chest-pain symptoms

CAD presentation	N	%	95% CI
Unstable angina (≤60 d)	1222	58.5	56.3–60.6
STEMI (≤7 d)	373	17.8	16.3–19.5
Stable angina (≤42 d)	244	11.7	10.4–13.1
NSTEMI (≤7 d)	98	4.7	3.9–5.7
NSTEMI (8–30 d)	72	3.4	2.7–4.3
STEMI (8–30 d)	42	2.0	1.5–2.7
Sx unlikely ischaemic (≤14 d)	24	1.1	0.8–1.7
No Sx / no angina (≤14 d)	15	0.7	0.4–1.2
Chest-pain symptoms			
Typical angina	1906	91.2	90.0–92.4
Atypical angina	140	6.7	5.6–7.8
Non-anginal chest pain	27	1.3	0.8–1.8
Asymptomatic	17	0.8	0.4–1.2

CAD, coronary artery disease; CI, confidence interval; NSTEMI, non-ST-segment elevation myocardial infarction; STEMI, ST-segment elevation myocardial infarction; Sx, symptoms; UA, unstable angina. The value in parentheses after each presentation denotes the time window (in days) between symptom onset and hospital presentation used to categorise the event (e.g., "≤7 d" = symptom onset within 7 days of presentation; "8–30 d" = onset 8 to 30 days before presentation). Proportions are reported with 95% Wilson confidence intervals.

Table-3: Procedural and angiographic characteristics

Characteristic	N	%	95% CI
Vessel disease (n = 1,932)			
Normal	291	15.1	13.5–16.7
Single	572	29.6	27.6–31.7
Double	593	30.7	28.7–32.8
Triple	476	24.6	22.8–26.6
PCI status (n = 1,371)			
Elective	1021	74.5	72.1–76.7
Emergency	236	17.2	15.3–19.3
Urgent	114	8.3	7.0–9.9
PCI indication (n = 1,371)			
Staged PCI	706	51.5	48.8–54.1
PCI for high-risk NSTEMI / unstable angina	346	25.2	23.0–27.6
PCI for STEMI (unstable, >12 h)	125	9.1	7.7–10.8
PCI for STEMI (stable, >12 h)	84	6.1	5.0–7.5
Primary PCI for STEMI	59	4.3	3.4–5.5
Other	20	1.5	0.9–2.2
PCI for STEMI (stable, post-thrombolysis)	18	1.3	0.8–2.1
Rescue PCI	13	0.9	0.6–1.6

CI, confidence interval; NSTEMI, non-ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction. Denominators differ because not all patients underwent every assessment: coronary angiography was performed in 1,932 patients (vessel-disease analysis), of whom 1,371 proceeded to PCI (status and indication analyses). Timing categories are defined as follows: primary PCI for STEMI = PCI within 12 h of symptom onset as the primary reperfusion strategy; PCI for STEMI (unstable, >12 h) = PCI performed more than 12 h after onset in a haemodynamically or electrically unstable patient; PCI for STEMI (stable, >12 h) = PCI more than 12 h after onset in a clinically stable patient; staged PCI = planned non-culprit revascularisation performed during a separate session. Proportions are reported with 95% Wilson confidence intervals.

Table-4: Cardiovascular medication uses after discharge (N = 2,090)

Medication	Prescribed at discharge, n (%)	95% CI
Aspirin	1987 (95.1%)	94.1–95.9
Clopidogrel	1977 (94.6%)	93.5–95.5
ACE inhibitor	1678 (80.3%)	78.5–81.9
Non-statin lipid therapy	3 (0.1%)	0.0–0.4

ACE, angiotensin-converting enzyme; CI, confidence interval. Proportions are reported with 95% Wilson confidence intervals.

Table-5a: Sex differences in heart rate (Welch's t-test)

Group	N	Mean	SD	MD	95% CI	t	*p*	d
Female	731	86.21	14.15	4.05	2.77–5.33	6.212	<0.001	0.284
Male	1359	82.16	14.36	—	—	—	—	—

MD, mean difference; SD, standard deviation; d, Cohen's d.

Table-5b: Systolic blood pressure across districts (one-way ANOVA)

District	N	Mean	SD	df	F	*p*	η^2
Charsadda	320	137.2	24.5	4, 2085	0.924	0.449	0.0018
Lower Dir	225	140.5	26.6				
Mardan	390	138.8	24.8				
Peshawar	868	139.3	24.8				
Other	287	140.6	22.6				

SD, standard deviation; df, degrees of freedom; η^2, eta-squared (effect size).

Table-5c: Predictors of length of stay (negative binomial regression)

Predictor	IRR	95% CI	*p*
(Intercept)	5.11	4.52–5.77	<0.001
Age (per SD)	1.03	0.98–1.08	0.250
Heart rate (per SD)	1.08	1.03–1.13	0.001
Diabetes mellitus	1.20	1.09–1.32	<0.001
Hypertension	1.15	1.05–1.26	0.004
Male sex	1.02	0.92–1.12	0.695
Killip III (vs I/II)	0.95	0.84–1.08	0.438
Killip IV (vs I/II)	1.05	0.94–1.17	0.425

CI, confidence interval; IRR, incidence rate ratio; SD, standard deviation. Model fit: AIC = 12,007.2; residual deviance = 362.3 on 2,082 degrees of freedom.

DISCUSSION

The multicenter analysis of 2,090 young adults admitted with cardiomyopathy indicates that there is a high number of modifiable cardiometabolic risk factors, that patients are already advanced in their disease, and that coronary anatomy is complicated. Hypertension (50%) and diabetes mellitus (38.7%) were seen in a population of just 38 years old with a median age of 38 years old, suggesting an early and aggressive cardiometabolic risk profile.

As previously discussed, the frequent co-existence of diabetes and hypertension is important. Chronic hyperglycaemia damages endothelial cells via increased oxidative stress, accumulation of advanced glycation end products, and lower nitric oxide bioavailability, whereas insulin resistance perpetuates an inflammatory and thrombotic state which encourages the formation of plaque and thrombosis.[11,12] Hypertension puts a high load on the heart, increases shear stress, and activates the renin–angiotensin–aldosterone system, which leads to arterial damage and left ventricular remodelling. These pathways may function together, resulting in this young South Asian population having premature and diffuse coronary disease.[22–24, 35]

A particularly concerning finding was the predominance of higher Killip classes, with 70% of patients in Class III–IV at admission. Beyond the tertiary-referral nature of the participating hospitals, this pattern is consistent with delayed presentation and limited access to timely care. Mechanistically, a higher Killip class reflects a larger ischaemic or infarcted myocardial mass and greater neurohormonal activation, both of which are linked to worse in-hospital outcomes.[25] The clinical picture was predominantly acute – unstable angina (58.5%) and STEMI being the leading presentations – mirroring patterns reported for acute coronary syndromes in developing regions and reinforced by current European Society of Cardiology and American Heart Association guidance, which emphasise that timely reperfusion, particularly early PCI, improves survival.[26,27]

Multivessel disease dominated the angiographic population with double- or triple-vessel involvement at 55.3% of patients, and 85% of those who were angiographically examined had at least some degree of obstructive disease. The morphology of the lesions was complex as 40.1% were classified as ACC/AHA Type C with a mean length of 20.9 mm. This is comparable to figures in several Western registries (e.g., e-Ultimate registry with a mean length of 27% of PCI features[33,34], and corresponds to the prevalence of South Asian patients with more extensive and early coronary disease[35]. Type C lesions such as tortuosity, severe calcification, chronic total occlusion had lower rates of success and more complications indicating the need for specialist interventional expertise and equipment.

The relatively high rate of staged PCI (51.5%) reflects a deliberately cautious revascularisation strategy for this complex anatomy – substantially higher than the approximately 14% staging rate in the SYNTAX trial, where staging was reserved largely for the most anatomically challenging cases or to limit contrast exposure and the risk of contrast-induced nephropathy. Adopting a staged approach for lesions averaging around 21 mm aligns with the COMPLETE trial, which demonstrated that staged complete revascularisation reduces major adverse cardiovascular events compared with culprit-only treatment in multivessel disease.[7,36] The predominance of typical angina (91.2%) alongside this procedural complexity supports the role of staged PCI for both symptom relief and risk reduction in this high-burden population.

We observed a modestly higher resting heart rate in women than in men (86.2 vs 82.2 bpm; Cohen's d = 0.28). Although the difference reached statistical significance in this large sample, its magnitude is clinically small and is consistent with well-described physiological differences in autonomic balance – women tend to have a relatively greater vagal-to-sympathetic ratio and intrinsic sinoatrial differences – rather than a marker of greater disease severity.[32] Together with the higher smoking

prevalence among men, these patterns reinforce the value of sex-specific preventive messaging.

Diabetes, hypertension, and admission heart rate were the dominant predictors of length of stay, whereas age and Killip class were not. The absence of an age effect is expected given the narrow eligibility window (18–45 years), which limits age variability and its ability to discriminate hospitalisation duration. The lack of an independent Killip effect – despite its established prognostic weight – suggests that early procedural and pharmacological management at these tertiary centres may have blunted the influence of admission haemodynamic class on overall stay. Hyperglycaemia may disrupt wound healing, the immune system, and endothelial recovery. An elevated heart rate upon admission may stimulate sympathetic responses and haemodynamic stress, both of which may delay recovery.[29–31] It is possible to alter these cardiometabolic risk factors through primary and secondary prevention, which could directly impact hospital length.

Adherence to guideline-directed therapy at discharge was reassuringly high for aspirin (95.1%), clopidogrel (94.6%), and ACE inhibitors (80.3%). The very low recorded use of statins and newer antiplatelet agents, however, points to a potential gap in comprehensive secondary prevention, possibly reflecting cost, availability, or documentation limitations.[28] Closing this gap is likely to be one of the most cost-effective ways to improve long-term outcomes in this cohort.

There are a few limitations of the study. The cross-sectional, observational design precludes causal inference, and the absence of long-term follow-up prevents assessment of downstream outcomes. The data were gathered through HMIS and hospital records, but cannot be guaranteed to be complete or correct, and some diagnoses may be based on administrative rather than clinical criteria. All hospitals in the study were tertiary-care, public-sector hospitals, which creates bias in referral and selection, as the patient population may be over-represented in severe conditions but under-represented in private or primary care or in one specific area of the country. Some estimates may have been affected by the inaccurate reporting of comorbidities and discharge medications. The study also has some strengths, including having a real-world multicenter cohort of young adults, robust statistical methods with confidence intervals, and detailed angiographic and procedural data. Further research should focus on prospective designs with long-term follow-up, independently confirmed diagnoses, and including primary care and private-sector patients, along with dedicated studies on preventive cardiology and early detection in this vulnerable group.

CONCLUSION

In this multicenter cohort of young adults with cardiovascular disease, presentation of modifiable risk factors was high, along with acute conditions, especially unstable angina and STEMI, advanced Killip class, and a high rate of multivessel disease and Type C lesions. Diabetes, hypertension, and elevated admission heart rate, rather than age or severity class, were the most common factors that led to prolonged hospitalisation. Adherence to the core guideline recommendations was high at discharge, but low use of statins and new antiplatelet agents suggested a lack of secondary prevention. Together, these data highlight the importance of earlier detection, increased control of modifiable risk factors, and prevention strategies to reduce the burden of cardiovascular disease among young adults.

AUTHORS' CONTRIBUTION

All authors contributed equally.

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