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Coronary Angiographic Patterns in Acute Coronary Syndrome Among Adults ≤ 45 Years and Their Relation to Clinical Risk Factors

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ABSTRACT

Background: Premature acute coronary syndrome (ACS) is rising rapidly in India, yet angiographic characterisation of patients under 45 years remains limited. Defining lesion distribution and its clinical correlation is essential for risk stratified prevention and revascularisation planning.

Methods: We undertook a prospective, single centre study of 46 consecutive adults (mean age 40.2 ± 4.0 years; 84.8 % men) admitted with STEMI, NSTEMI or unstable angina between January 2019 and December 2021. All underwent coronary angiography (CAG) within 48 hours. CAG characteristics were studies. Associations with demographic, metabolic (HbA1c, lipid profile) and inflammatory markers (high sensitivity C reactive protein [hsCRP]) were tested using χ^2 or Fisher exact tests ($\alpha = 0.05$).

Results: Obstructive CAD was present in all patients. SVD predominated (65.2 %), half of which involved the proximal left anterior descending artery (LAD). DVD and TVD accounted for 8.7 % and 10.9 %, respectively, while left main disease was uncommon (6.5 %). Intracoronary thrombus was visualised in 21.7 % and correlated with hsCRP > 1 mg/dl ($p = 0.02$). Multivessel disease clustered with occult diabetes (HbA1c ≥ 6.5 %; odds ratio [OR] 3.4, 95 % CI 1.1–11.0; $p = 0.03$) and elevated LDL cholesterol ≥ 130 mg/dl (OR 2.9, 95 % CI 1.0–8.7; $p = 0.04$). Angiographic calcification was absent.

Conclusion: Young Indian adults with ACS typically harbour focal, thrombus rich proximal LAD lesions, whereas multivessel involvement clusters with unrecognised diabetes and high LDL cholesterol. Early cardiometabolic screening, aggressive lipid lowering may prevent diffuse atherosclerosis and improve long term outcomes in this vulnerable population.

Keywords: *Young adults, Acute coronary syndrome, Myocardial infarction, Unstable angina, coronary angiography, Thrombus. Single vessel disease, Multivessel disease.*

INTRODUCTION

Coronary artery disease (CAD) is no longer an affliction restricted to middle-aged and elderly populations. Over the past two decades, hospital registries across the globe have recorded a steady rise in the proportion of patients with acute coronary

syndrome (ACS) who are younger than 45 years (1,2). The early loss of productive life-years and the attendant socioeconomic impact give premature CAD an urgency that exceeds its absolute numerical burden. Yet, despite the mounting public-health importance, the anatomic substrate and biological drivers of early-onset ACS remain incompletely characterised.

Large Western registries such as the NRMI-2 and GRACE programmes have consistently shown that the angiographic footprint of ACS in the young differs from that in older patients (3). Whereas multivessel atherosclerosis and heavy calcification dominate in late-middle-aged adults, younger patients more often exhibit discrete, soft plaques with high thrombotic propensity but limited overall plaque load culminating in a high prevalence of single-vessel disease (SVD). Proximal left-anterior-descending (LAD) segments are over-represented, a pattern attributed to complex interactions between shear stress, turbulent flow and lipid-core composition (4). Such observations, however, stem primarily from North-American and European cohorts; whether they apply to South-Asian patients who possess a unique cardiometabolic milieu characterised by visceral adiposity, insulin resistance and hyper-apoB dyslipidaemia despite "normal" body-mass indices (5).

Adding further complexity, genetic variants affecting lipoprotein(a), apolipoprotein E, and haptoglobin phenotypes, all of which influence atherothrombosis, demonstrate population heterogeneity (6). Polymorphisms in the 9p21 locus, for example, are more prevalent among Indians and have been implicated in both plaque progression and rupture susceptibility (7). Environmental exposures ranging from early childhood under-nutrition to air-particulate pollution and chronic psychosocial stress feed into a "double-hit" paradigm, whereby accelerated endothelial ageing primes coronary arteries for atherosclerosis that unravels under the inflammatory insult of adulthood (8). Smoking remains rampant among young men, amplifying oxidative stress and shifting the thrombotic–fibrinolytic balance towards occlusion (9). Meanwhile, poorly screened glycaemic perturbations lead to glycoxidative plaque vulnerability even before frank diabetes is diagnosed.

Against this multifactorial backdrop, the detailed angiographic anatomy of young Indian ACS patients has not been systematically documented. Most available data derive from small, single-centre series without uniform imaging protocols or contemporary definitions of culprit-lesion morphology. Consequently, the true frequency of proximal LAD involvement, the burden of multivessel disease, and the prevalence of angiographic thrombus or non-atherosclerotic substrates such as coronary artery spasm, spontaneous dissection or congenital anomalies remain uncertain. These knowledge gaps hamper risk stratification and the design of targeted primary-prevention strategies.

Understanding lesion distribution is clinically relevant for several reasons. First, proximal LAD occlusion often labelled the "widow-maker" portends larger infarct size, lower left-ventricular ejection fraction and worse long-term prognosis if reperfusion is delayed (10). Second, the need for percutaneous intervention versus coronary bypass grafting hinges on whether disease is confined to a single epicardial vessel or involves multiple territories. Third, anatomical patterns may provide indirect clues to the pathobiology at play: plaque rupture typically yields red-thrombus-rich lesions in high-shear segments, whereas plaque erosion manifests as white-thrombus and is more common in smokers and women (11). Finally, establishing the angiographic phenotype of young CAD could inform recommendations on the use of advanced imaging (e.g., coronary CT angiography) for asymptomatic relatives, thereby enabling truly preventive cardiology.

Several investigators have postulated that Indians present a paradox: despite possessing a heavy systemic inflammatory burden as reflected by high-sensitivity C-reactive protein (hsCRP) levels they tend to harbour focal coronary lesions rather than diffuse disease (12,13). A plausible explanation is that heightened inflammation accelerates the transition from lipid-rich fatty streaks to rupture-prone thin-cap fibro-atheromas in a limited number of haemodynamically stressed sites, precipitating clinical events before the widespread progression of atherosclerosis. If confirmed, such a paradigm would underscore the promise of early aggressive lipid-lowering and anti-inflammatory therapies interventions that could arrest lesion evolution at a phase amenable to medical reversal.

To address these uncertainties, we analysed coronary angiograms of adults ≤45 years presenting with ACS to a tertiary care hospital in Mumbai over a two-year period. By correlating angiographic patterns (vessel involvement, segment location, presence of thrombus, and extent of disease) with clinical, biochemical and inflammatory profiles, we aimed to elucidate (i) the dominant culprit-lesion anatomy, (ii) the frequency and predictors of multivessel versus single-vessel disease, and

(iii) the relationship between systemic inflammation and intracoronary thrombus burden. We hypothesised that most young Indian ACS patients would exhibit isolated proximal LAD SVD, and that metabolic dysregulation particularly occult diabetes and elevated LDL-cholesterol would segregate with multivessel disease. Our findings seek to enrich the limited Indian literature on premature CAD, provide a foundation for future multicentre registries, and stimulate discussion on tailored prevention and management algorithms for this vulnerable demographic.

In the sections that follow, we outline the study design and quantitative coronary-analysis methods employed, present detailed results stratified by lesion pattern, and critically appraise the implications of our observations in light of existing global evidence. By delineating the anatomic signature of early-onset ACS in an urban Indian cohort, we hope to contribute a small but essential piece to the larger puzzle of why and how coronary arteries fail at such a young age.

MATERIALS AND METHODS

Study design and setting

We performed a prospective, observational study at the Department of Cardiology in a Tertiary-Care Hospital, in western India. Consecutive adults ≤ 45 years of age who presented with acute coronary syndrome (ACS) between 1 January 2019 and 31 December 2021 were screened for eligibility.

Participants

Between 1 January 2019 and 31 December 2021 we prospectively enrolled forty-six consecutive adults aged ≤ 45 years who presented to a Tertiary-Care Hospital, in western India with a working diagnosis of acute coronary syndrome (ACS). Eligibility required fulfilment of at least one component of the Fourth Universal Definition of Myocardial Infarction, namely, (i) ischaemic chest pain or angina-equivalent symptoms lasting ≥20 minutes, (ii) new or presumed-new ECG evidence of myocardial ischaemia (ST-segment deviation ≥ 1 mm or new left-bundle-branch block), or (iii) a rise and/or fall in high-sensitivity cardiac-troponin-T above the 99th-percentile upper-reference limit. Patients were excluded if they had a history of prior coronary revascularisation, congenital structural heart disease, myocarditis or pericarditis, spontaneous coronary artery dissection, stage ≥3 chronic kidney disease, systemic autoimmune or inflammatory disorders, or if they declined written informed consent. All eligible participants underwent comprehensive clinical evaluation, laboratory testing and diagnostic coronary angiography within 48 hours of admission.

Sample size

Using an anticipated single-vessel disease (SVD) prevalence of 60 % in young ACS (from pilot data) with 15 % absolute precision and 95 % confidence, the calculated minimum sample was 42; we enrolled 46 patients.

Clinical and biochemical data collection

A structured case-record form captured demographics, anthropometrics, cardiovascular risk factors, and medication history. Blood samples (fasting when feasible, otherwise within 24 h of admission) were analysed for:

- fasting plasma glucose and HbA1c (high-performance liquid chromatography);
- lipid profile (enzymatic colorimetry);
- high-sensitivity C-reactive protein (hsCRP; immunoturbidimetric assay).

Definitions

- Diabetes mellitus: history of diabetes or HbA1c ≥ 6.5 %.
- Hypertension: documented diagnosis, antihypertensive use, or office BP ≥ 140/90 mm Hg.
- Dyslipidaemia: any of LDL-C ≥ 130 mg/dl, HDL-C < 40 mg/dl (men) / < 50 mg/dl (women), triglycerides ≥ 150 mg/dl, or statin use.
- Obesity: body-mass index (BMI) ≥ 30 kg/m².

Coronary angiography and lesion assessment

All patients underwent invasive coronary angiography within 48 h of admission by the radial or femoral approach using 5 Fr or 6 Fr Judkins catheters. Quantitative coronary analysis (QCA) was performed offline by two experienced interventional cardiologists blinded to clinical data; discrepancies > 5 % diameter stenosis were adjudicated by a third reader.

- Significant stenosis: luminal diameter reduction ≥ 50 % in any epicardial vessel (reference diameter ≥ 2.0 mm).
- Disease extent:
  - SVD: ≥1 significant lesion in one major epicardial vessel;
  - DVD/TVD: lesions in two or three vessels;
  - Left-main disease: ≥50 % stenosis in the left main coronary artery (LMCA).
- Lesions were coded by American Heart Association (AHA) 15-segment model; the culprit-lesion was identified by angiographic appearance (fresh thrombus, ulceration, flow limitation) and ECG correlation.
- Intracoronary thrombus was diagnosed when a discrete, filling-defect, contrast-dammed image was contiguous with the vessel wall on two orthogonal views.

Statistical analysis

Data were analysed with IBM SPSS Statistics v26.0. Continuous variables are expressed as mean ± SD (if normally distributed) or median [IQR]; categorical variables as counts (percentages). Group comparisons employed the independent-samples t-test or Mann-Whitney U-test for continuous data and χ^2 test or Fisher's exact test for categorical data. Odds ratios (ORs) with 95 % confidence intervals (CIs) were calculated for associations between multivessel disease and risk factors. A two-sided $p < 0.05$ denoted statistical significance.

Ethical considerations

The study protocol conformed to the Declaration of Helsinki (2013 revision) and received approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enrolment.

RESULTS

Overall angiographic burden

All 46 participants with ACS underwent diagnostic coronary angiography within 48 hours of admission. Obstructive coronary artery disease (CAD) was observed in all patients. Single-vessel disease (SVD) predominated, affecting 30/46 cases (65.2 %); double-vessel disease (DVD) and triple-vessel disease (TVD) were far less frequent at 8.7 % and 10.9 %**,** respectively, and significant left-main coronary artery (LMCA) stenosis was observed in 3 patients (6.5 %)**.** The baseline cardiometabolic risk factors and the extent of coronary disease is depicted in Table 1.

Vessel- and segment-wise distribution

The left-anterior-descending artery (LAD) was by far the commonest culprit, being involved in 33 patients (71.7 %); half of these lesions lay in the proximal segment (23/46, 50 %) . Right-coronary-artery (RCA) disease was next most frequent (18/46, 39.1 %), followed by the left-circumflex (LCx) and major diagonal (D) branches (each 11/46, 23.9 %). LMCA disease, posterior-descending artery (PDA) involvement and ramus/obtuse-marginal lesions were all uncommon (≤6.5 %) (Table 2).

Other angiographic features

Fresh intracoronary thrombus was visualised in 10 patients (21.7 %), while a systolic myocardial bridge over the mid-LAD was noted in 2 patients (4.3 %); no angiographic calcification, dissection or anomalous origin was detected.

Clinical presentation and laboratory correlates

Clinically, NSTEMI was the commonest mode of presentation (19/46, 41.3 %), followed by STEMI (18/46, 39.1 %) and unstable angina (9/46, 19.6 %) (Table 3). Raised high-sensitivity C-reactive protein (hsCRP > 1 mg/dl) was present in 38/46 patients (82.6 %) and was the only variable that showed a statistically significant association with ACS phenotype ($p < 0.001$).

TABLES

Table 1. Baseline cardiometabolic risk factors according to extent of coronary disease (BMI: Body Mass Index; HbA1C: Glycated Haemoglobin; hs-CRP: High sensitivity C Reactive Protein; LDL-C: Low density lipoprotein cholesterol)

Risk factor	Single (n = 37)	Multi-vessel (n = 9)	*p*-value
Current smoker, n (%)	17 (45.9)	6 (66.7)	0.31
Hypertension, n (%)	9 (24.3)	2 (22.2)	1.00
Obesity (BMI ≥ 30 kg/m), n (%)	18 (48.6)	6 (66.7)	0.31
HbA1c ≥ 6.5 %, n (%)	18 (48.6)	7 (77.8)	0.03
LDL-C ≥ 130 mg/dl, n (%)	18 (48.6)	8 (88.9)	0.04
hsCRP > 1 mg/dl, n (%)	29 (78.4)	9 (100)	0.11

Table 2. Vessel-wise distribution of significant coronary lesions (N = 46)

Coronary segment	Patients affected n (%)
Left-main coronary artery (LMCA)	3 (6.5)
Left-anterior-descending (LAD)	33 (71.7)
Left-circumflex (LCx)	11 (23.9)
Right coronary artery (RCA)	18 (39.1)
Major diagonal (D)	11 (23.9)
Posterior-descending artery (PDA)	3 (6.5)

Table 3. Acute-coronary-syndrome (ACS) phenotype by extent of coronary disease (STEMI: ST elevation myocardial infarction, NSTEMI: Non-ST elevation myocardial infarction)

ACS presentation	Single (n = 37)	Multi-vessel (n = 9)	*p*-value
STEMI, n (%)	14 (37.8)	4 (44.4)	0.92
NSTEMI, n (%)	16 (43.2)	3 (33.3)	—
Unstable angina, n (%)	7 (18.9)	2 (22.2)	—

Table 4. Intracoronary thrombus in relation to systemic inflammation (hsCRP)

hsCRP category	Thrombus present n / N (%)	*p*-value
≤ 1 mg/dl	0 / 8 (0.0)	0.02
> 1 mg/dl	10 / 38 (26.3)	—

DISCUSSION

The present study provides one of the most granular angiographic characterisations yet reported for Indian adults ≤45 years presenting with acute coronary syndrome (ACS). Our principal observations, namely, the pre-eminence of single-vessel disease (SVD) centred on the proximal left-anterior-descending artery (LAD); the clustering of multivessel involvement with occult diabetes and elevated LDL-cholesterol; and the strong association between systemic inflammation (hsCRP > 1 mg/dl) and intracoronary thrombus carry important pathogenetic and clinical implications (Table 4). They also confirm and extend patterns described in Western and pan-Asian cohorts while underscoring several India-specific nuances.

First, the dominance of focal proximal LAD lesions (71.7 % of all culprit vessels, half in the proximal segment) mirrors data from the YOUNG-MI registry in the United States, where 63 % of events in patients <50 years involved the LAD (14). Haemodynamic shear stress, flow separation and oscillatory wall stress within the proximal LAD are thought to accelerate endothelial dysfunction and lipid deposition, culminating in thin-cap fibro-atheromas prone to rupture (15,16). South-Asian individuals may be particularly susceptible owing to intrinsic endothelial nitric-oxide-synthase polymorphisms and heightened oxidative stress even at ostensibly "normal" body-mass indices which drive premature plaque vulnerability (17). The near-absence of angiographic calcification in our cohort further substantiates the hypothesis that plaque rupture rather than progressive calcific narrowing predominates in young patients. Lack of calcification has practical ramifications: calcium-free culprit lesions are technically easier to treat with drug-eluting stents, but the absence of stable fibro-calcific rings may also facilitate late positive remodelling and in-stent restenosis, mandating meticulous secondary prevention.

Second, although SVD accounted for two-thirds of cases, multivessel disease (double or triple) was not trivial (19.6 %) and was tightly linked to previously undiagnosed diabetes (HbA1c ≥ 6.5 %) and high LDL-C. This echoes observations from Jamal Rana et al (2025) which reported a three- to four-fold higher probability of multivessel disease in diabetic young adults (18). Chronic hyperglycaemia enhances non-enzymatic glycation of LDL particles, increasing their arterial retention and fostering diffuse intimal lipid pools (19). Our data therefore support routine HbA1c screening in ostensibly nondiabetic Indian youth with ACS symptoms and argue for aggressive LDL-lowering potentially to <55 mg/dl, as mandated by recent ESC guidelines for "very high-risk" individuals (20). The finding that 88.9 % of multivessel cases harboured LDL-C ≥ 130 mg/dl yet only 22 % were on statin-therapy at presentation highlights a missed preventive opportunity.

Third, systemic inflammation emerged as a pervasive theme: 82.6 % of patients had hsCRP > 1 mg/dl, and elevated hsCRP was the single strongest correlate of angiographically visible thrombus. This aligns with the CANTOS trial's demonstration that interleukin-1β blockade reduces recurrent events disproportionately in patients with residual inflammatory risk (21). Inflammation promotes tissue factor expression, platelet activation and fibrin-rich "red" thrombus, explaining our 26.3 % thrombus rate among those with high hsCRP versus 0 % when hsCRP was low. From a management standpoint, such thrombus-rich lesions may benefit from tailored antithrombotic strategies e.g., prolonged Cangrelor use or thrombus aspiration although randomised evidence in young cohorts remains scant (22). Moreover, lifestyle modifiers of inflammation (weight loss, aerobic exercise, cessation of smoking and particulate exposure) should be emphasised in secondary prevention programmes.

Fourth, Only 15.2 % of our cohort were female, that reinforces growing recognition that young women remain under-represented in ACS registries yet experience worse outcomes; study by Benamer et al suggests women suffer longer

delays to reperfusion and higher in-hospital mortality (23). While our sample size precludes sex-specific conclusions, future multicentre registries must oversample women to elucidate hormonal and microvascular contributors to premature CAD. Comparison with domestic literature is instructive. The Kerala ACS Registry reported SVD in 58 % of patients ≤40 years nearly identical to our 65 % but a higher prevalence of RCA involvement (24). Dietary patterns (high coconut-oil intake) and different genetic ancestry may underlie this discrepancy. Conversely, a study from central India showed LAD dominance (> 67.3 %) (25). Such geographical heterogeneity cautions against simplistic extrapolation and underscores the need for all-India prospective consortia.

Several limitations merit acknowledgement. The study is single-centre and relatively small, raising the possibility of selection bias; however, consecutive enrolment, rigorous angiographic adjudication and homogeneous imaging protocols mitigate this concern. Second, absence of intravascular ultrasound or optical coherence tomography precluded detailed plaque-morphology assessment. Third, we did not assay lipoprotein(a) or apolipoprotein B, markers known to modulate premature atherothrombosis; ongoing analyses incorporating these parameters will refine risk stratification. Fourth, hsCRP was measured only once; serial measurements could clarify whether persistent versus transient inflammation drives recurrent events. Finally, the observational design prevents causal inference—yet the biologic plausibility and consistency with external datasets lend credence to the associations observed.

In aggregate, our findings crystallise a dual-pathway model of premature ACS in Indian youth: a majority experience focal proximal LAD rupture against a backdrop of systemic inflammation and smoking, whereas a metabolically driven subgroup, burdened by occult diabetes and elevated LDL-C, progresses to diffuse multivessel disease. This dichotomy suggests complementary preventive strategies: primordial prevention targeting tobacco and inflammation in the general population, and intensive lipid/ glycaemic control in metabolically predisposed individuals identified through school- and workplace-based screening. Furthermore, the high thrombus burden among patients with raised hsCRP hints at potential benefit from longer dual-antiplatelet therapy or novel anti-inflammatory agents, hypotheses that warrant testing in randomised trials focused exclusively on young ACS.

In conclusion, our study bridges a critical knowledge gap by detailing the angiographic substrate of ACS in Indian adults ≤45 years and linking distinct lesion patterns to modifiable risk factors. By integrating these insights into public-health policy and clinical practice from early statin initiation to community-level smoking cessation India can hope to stem the tide of cardiovascular events striking its youth at the prime of life.

CONCLUSION

In this cohort, two-thirds of young ACS patients harboured isolated proximal LAD disease, whereas multivessel and left-main involvement, though infrequent, clustered with occult diabetes and high LDL-C. Routine cardiometabolic screening in adolescents and young adults, coupled with prompt revascularisation of culprit lesions, may avert transition to diffuse coronary atherosclerosis. Future multicentre registries should explore genetic and inflammatory signatures driving these patterns.

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