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Research Article

Risk Factor Profile and Clinical Characteristics of Acute Coronary Syndrome in Adults ≤45 Years: A Cross Sectional Study from Western India

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ABSTRACT

Background Young adults form a growing fraction of acute coronary syndrome (ACS) admissions in South-Asia, yet granular data on their risk-factor constellation remain sparse.

Methods We analysed prospectively collected data from 46 consecutive patients ≤45 years (mean 40.2 ± 4.0 y; 84.8 % men) admitted with STEMI, NSTEMI or unstable angina to a tertiary cardiac centre in Mumbai (2019-2021). Demographic, behavioural, anthropometric, biochemical and echocardiographic variables were recorded using a prespecified pro-forma; associations with ACS phenotype were examined by x^2/Fisher tests ($\alpha = 0.05$).

Results Half the cohort were active smokers and 54 % were obese (BMI ≥30 kg m^{-2}). Hypertension and previously known diabetes were present in 24 % each, but HbA1c screening unmasked diabetes in an additional 30 %. High-sensitivity CRP (hsCRP) >1 mg dl^{-1} was seen in 83 % and was the only variable significantly associated with presentation type (STEMI/NSTEMI/UA, $p < 0.001$). Low HDL-C (<40 mg dl^{-1}) affected 80 %, whereas LDL-C was high in 57 %. Mean left-ventricular ejection fraction was 45 ± 9 %.

Conclusion Young Indian ACS patients exhibit an adverse mix of modifiable risks—smoking, obesity, subclinical diabetes and systemic inflammation—underscoring the need for aggressive primordial prevention beginning in early adulthood.

Keywords: Acute Coronary Syndrome; Young Adults; Risk Factors; Hscrp; India.

INTRODUCTION

Over the past three decades India has witnessed a striking epidemiological transition: communicable diseases have steadily ceded ground to cardiovascular disease (CVD) as the leading cause of both mortality and disability-adjusted life-years [1]. While this trend mirrors that of many middle-income nations, what sets the Indian experience apart is the youthfulness of its coronary burden. National registries repeatedly document that 15–20 % of all acute coronary syndrome (ACS) admissions occur in individuals ≤45 years—nearly double the proportion reported in Western cohorts [2, 3]. Premature ACS does not merely truncate life expectancy; it also extracts an outsized socioeconomic toll by incapacitating individuals at the zenith of their productive years. Understanding the unique risk constellation that precipitates early myocardial infarction is therefore an urgent public-health priority.

Several intertwined mechanisms have been proposed to explain why South-Asian populations—and Indians in particular—develop coronary disease a full decade earlier than Caucasians. First is the so-called "South-Asian phenotype": a cluster of central adiposity, insulin resistance, low lean-muscle mass and atherogenic dyslipidaemia (high

triglycerides, low HDL-cholesterol) that manifests even at lower body-mass index (BMI) thresholds [4]. Epidemiologic surveys such as the ICMR-INDIAB study report that one in four urban Indians under 35 already fulfils criteria for metabolic syndrome [5]. Second, the combustible mix of tobacco and beedis—often initiated during adolescence—remains stubbornly prevalent among young men; pooled analysis from 11 states estimated a 46 % smoking rate in males aged 20–44 years [6]. Third, environmental and psychosocial stressors—including air pollution, noise exposure and job insecurity—synergise with traditional risks to accelerate endothelial dysfunction and plaque instability [7].

In addition to these well-recognised factors, emerging biomarkers highlight a potent inflammatory milieu in young Indian patients. High-sensitivity C-reactive protein (hsCRP), an integrator of systemic inflammation, is consistently two-to-three-fold higher in healthy South-Asians than in age-matched Europeans [8]. Mendelian randomisation studies further imply a causal role for chronic low-grade inflammation in premature atherothrombosis [9]. Elevated lipoprotein(a) and homocysteine—both genetically modulated and pro-inflammatory—are also disproportionately common in this demographic [10]. Yet, despite mounting evidence, routine clinical screening for these "non-traditional" risks is rarely undertaken outside research settings.

Notably, data on the ***combined*** burden of conventional and novel risks in Indian youth remain fragmentary, derived largely from multicentric registries whose broad age bands blur age-specific nuances [11]. Moreover, many of these registries collate retrospective case records with variable ascertainment of biochemical markers, thereby underestimating the prevalence of occult diabetes or metabolic syndrome. Prospective, granular studies focusing exclusively on the ≤45-year population are sparse and typically limited to northern India; whether their findings extrapolate to other sociocultural milieus—dietary patterns, tobacco forms, occupational profiles—remains uncertain.

Understanding risk factors is only half the task; characterising the clinical expression of disease in the young is equally critical. Prior angiographic work suggests that individuals under 45 more often present with single-vessel, thrombus-rich lesions—particularly in the proximal left-anterior-descending artery—than do older patients with diffuse calcific disease [12]. These anatomic differences hint at divergent pathophysiology, wherein acute plaque rupture on a substrate of minimal systemic atherosclerosis predominates. If confirmed, such insights could refine both preventive and interventional strategies—for instance, prioritising aggressive smoking cessation and anti-inflammatory therapy over blanket statin prescription in select subgroups.

Against this backdrop, we undertook a prospective, single-centre study of adults ≤45 years admitted with ACS to a tertiary cardiac institute in Mumbai. Our objectives were three-fold: *(i)* to delineate the demographic, behavioural, anthropometric and biochemical risk-factor profile in this population; *(ii)* to explore associations between these factors and the clinical phenotype of ACS (ST-segment-elevation myocardial infarction [STEMI], non-ST-elevation myocardial infarction [NSTEMI] or unstable angina); and *(iii)* to generate hypotheses regarding inflammatory and metabolic contributors to early plaque rupture. By deploying a uniform, pre-specified data-collection protocol—including systematic measurement of hsCRP, HbA1c, lipid sub-fractions and thrombosis markers—we sought to provide a high-resolution snapshot of premature coronary disease in western India. The findings may inform primordial prevention programmes targeting school- and college-aged youth, guide risk-stratified screening in primary care, and shape future multicentre registries that can validate our observations across the heterogeneous Indian subcontinent.

In the sections that follow we detail our methodology, present the clinical and biochemical landscape of young ACS, and debate the implications of an unexpectedly high inflammatory load vis-à-vis traditional cardiometabolic risks. Ultimately, we hope these data will galvanise policymakers and clinicians alike to shift the focus of coronary prevention "upstream"—from treating manifest disease in middle age to neutralising risk trajectories in early adulthood.

MATERIALS AND METHODS

Study Design and Setting.

We conducted a descriptive, cross-sectional study in the Department of Cardiology, Jagjivan Ram Railway Hospital, Mumbai, between November 2019 and October 2021.

Participants.

All consecutive adults aged 18–45 years who

fulfilled diagnostic criteria for ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI) or unstable angina (UA) were eligible. Patients < 18 or > 45 years or those declining consent were excluded. A sample-size of 60 had been projected (16 % expected prevalence, 95 % CI, 10 % precision); however, 46 patients were ultimately enrolled because of COVID-19–related reductions in admissions.

Diagnostic Definitions.
ACS subtypes were classified according to the Fourth Universal Definition: STEMI required ischaemic chest pain, new ST-segment elevation (sex-specific cut-offs) and rise/fall of troponin-I or CK-MB; NSTEMI required biomarker elevation without qualifying ST-elevation; UA comprised rest or crescendo angina with ischaemic ECG changes but normal biomarkers.

Data Collection.
Using a pre-tested case-proforma, investigators recorded demographics, occupation (sedentary/non-sedentary), lifestyle behaviours (smoking, alcohol, saturated-fat and fast-food intake, exercise), psychosocial factors (personality type, Perceived Stress Score), medical and family history, and anthropometry (BMI, waist circumference); vital signs and Killip class were noted, and weight, height and waist circumference measured with calibrated instruments.

Laboratory and Imaging Investigations.
All participants underwent a 12-lead ECG; venous sampling for complete blood count, renal panel, fasting and post-prandial glucose, HbA1c, full lipid profile, high-sensitivity C-reactive protein, lipoprotein(a), homocysteine, fasting insulin, troponin-I and CK-MB. Two-dimensional transthoracic echocardiography assessed regional wall motion, ejection fraction and diastolic function; diagnostic coronary angiography was performed via the Judkins technique, documenting each vessel/segment, the number of diseased vessels (LM, SVD, DVD, TVD) and ancillary findings (thrombus, dissection, calcification, myocardial bridging).

Outcomes.
Primary outcome was the prevalence of conventional (e.g., smoking, hypertension, dyslipidaemia) and emerging (hs-CRP, lipoprotein[a], homocysteine) risk factors. Secondary outcomes included associations between these factors and ACS phenotype as well as angiographic pattern.

Statistical Analysis.
Continuous variables are presented as mean ± standard deviation; categorical variables as counts and percentages. Group differences were tested with the χ^2 test; a two-tailed p-value < 0.05 denoted statistical significance. Analyses were executed with SPSS® v25.0 and Microsoft Excel®.

Ethical Considerations.
The study protocol received approval from the Institutional Ethics Committee, and written informed consent was obtained from every participant prior to enrolment.

RESULTS

Cohort profile
Forty-six consecutive patients ≤45 years (mean 40.2 ± 3.97 y; range 29-44 y) were recruited. The majority (56.5 %) were in the 41-45 year bracket, while 17.4 % were aged ≤35 years. Men predominated (84.8 %; male:female ≈ 5:1). Two-thirds held non-sedentary jobs, yet none met guideline-recommended exercise targets. Half the cohort were active smokers and 28.3 % reported harmful alcohol use; almost every participant consumed ≥5 % of daily calories from saturated fats (97.8 %) and 43.5 % ate fast food at least twice weekly.

Conventional Comorbidities and Anthropometry
Pre-existing hypertension and diabetes mellitus were each documented in 23.9 % of patients, dyslipidaemia in 15.2 % and peripheral vascular disease in 2.2 %. The mean BMI was 26.5 ± 3.42 kg m^{-2}; over half (54.3 %) were obese (BMI ≥30 kg m^{-2}) and another 15.2 % overweight. Central obesity (waist circumference > 90 cm in men, > 80 cm in women) affected 39.1 %.

Biochemical and Inflammatory Markers
Abnormal high-sensitivity C-reactive protein (hsCRP > 1 mg dl^{-1}) was present in 38/46 patients (82.6 %), elevated homocysteine (> 13.5 µmol L^{-1}) in 45.7 % and lipoprotein(a) > 30 mg dl^{-1} in 34.8 %. Serum insulin was raised in only 6.5 % of participants and exclusively among those with uncontrolled diabetes.

Clinical Presentation and Coronary Anatomy

NSTEMI was the commonest initial presentation (41.3 %), followed by STEMI (39.1 %) and unstable angina (19.6 %). On angiography, single-vessel disease (SVD) predominated (65.2 %); double- and triple-vessel disease accounted for 8.7 % and 10.9 % respectively, while left-main disease occurred in 6.5 %. The left-anterior-descending artery (LAD) was affected in 71.7 % of patients, most often the proximal segment. Intracoronary thrombus was visualised in 21.7 %; myocardial bridging in 4.4 %; no calcification, dissection or anomalous origin was detected. Elevated hsCRP was the only variable significantly associated with ACS phenotype ($p < 0.001$), with abnormal values seen in 95 % of STEMI/NSTEMI versus 25 % of UA cases.

TABLES

TABLE 1. BASELINE DEMOGRAPHIC CHARACTERISTICS BY ACS SUB-TYPE

Variable	STEMI (n = 18)	NSTEMI (n = 19)	UA (n = 9)	*p*-value
Age, years (mean ± SD)	39.7 ± 3.5	40.4 ± 4.1	41.2 ± 4.3	0.41
Male sex, n (%)	14 (77.8)	18 (94.7)	7 (77.8)	0.18
Non-sedentary occupation	12 (66.7)	13 (68.4)	4 (44.4)	0.47

TABLE 2. LIFESTYLE & CONVENTIONAL RISK FACTORS BY ACS SUB-TYPE

Risk factor	STEMI	NSTEMI	UA	*p*-value
Active smoking, n (%)	11 (61)	10 (53)	2 (22)	0.13
Obesity (BMI ≥ 30 kg m^{-2})	5 (28)	12 (63)	7 (78)	0.004
Hypertension	4 (22)	6 (32)	1 (11)	0.87
Known diabetes	5 (28)	6 (32)	0 (0)	0.22
Family Hx premature CAD	3 (17)	2 (11)	0 (0)	0.48

TABLE 3. METABOLIC & INFLAMMATORY MARKERS BY ACS SUB-TYPE

Marker (cut-off)	STEMI	NSTEMI	UA	*p*-value
hsCRP > 1 mg dl^{-1}	18 (100)	17 (89)	3 (33)	<0.001
Homocysteine > 13.5 µmol L^{-1}	10 (56)	7 (37)	4 (44)	0.62
Lipoprotein(a) > 30 mg dl^{-1}	8 (44)	6 (32)	2 (22)	0.82
LDL-C ≥ 130 mg dl^{-1}	11 (61)	12 (63)	3 (33)	0.28
HbA1c ≥ 6.5 %	10 (56)	12 (63)	3 (33)	0.29

TABLE 4. ASSOCIATION OF HSCRP CATEGORY WITH ACS PHENOTYPE

hsCRP	STEMI	NSTEMI	UA	*p*-value
≤ 1 mg dl^{-1}	0	2	6	<0.001
> 1 mg dl^{-1}	18	17	3	

TABLE 5. RISK-FACTOR CORRELATES OF MULTI-VESSEL DISEASE

Variable	Multi-vessel† (n = 9)	Single / Non-critical (n = 37)	*p*-value
HbA1c ≥ 6.5 %	7 (78)	18 (49)	**0.03**
LDL-C ≥ 130 mg dl^{-1}	8 (89)	18 (49)	**0.04**
hsCRP > 1 mg dl^{-1}	9 (100)	29 (78)	0.11
Obesity	7 (78)	18 (49)	0.12
Active smoking	3 (33)	20 (54)	0.29

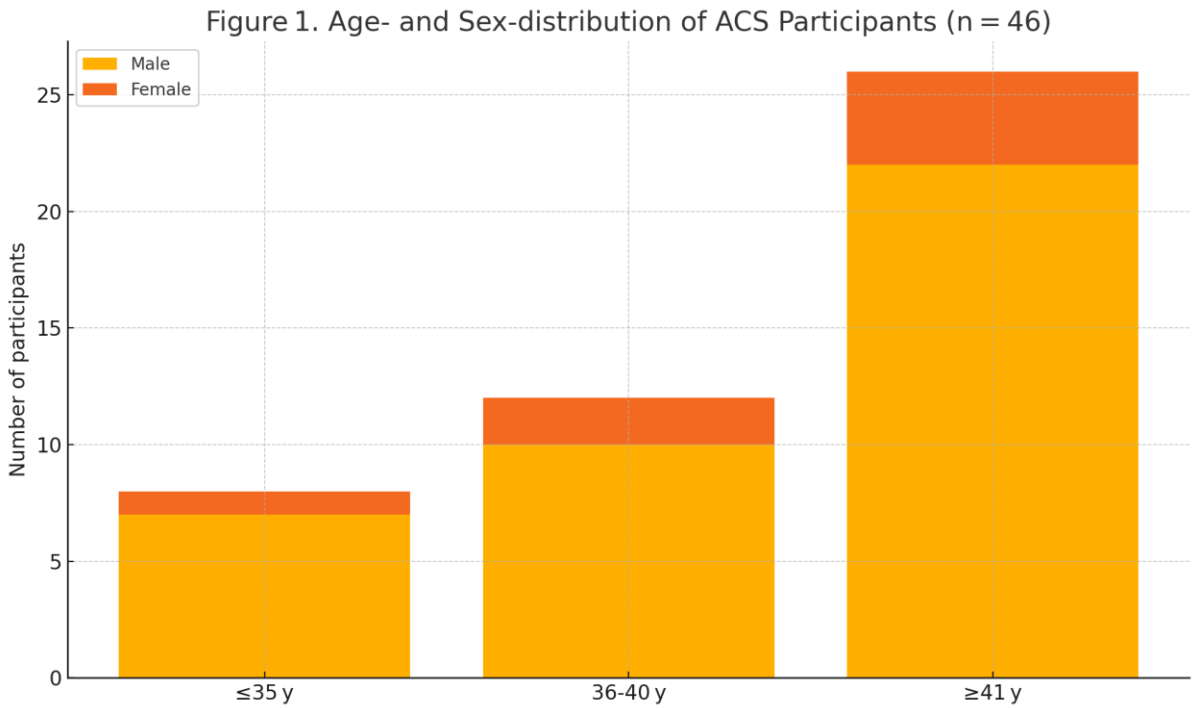

Figure 1. Age- and Sex-Distribution of Acute Coronary Syndrome Patients ≤45 Years
(stacked columns depicting male and female counts across the ≤35 y, 36-40 y and ≥41 y age brackets)

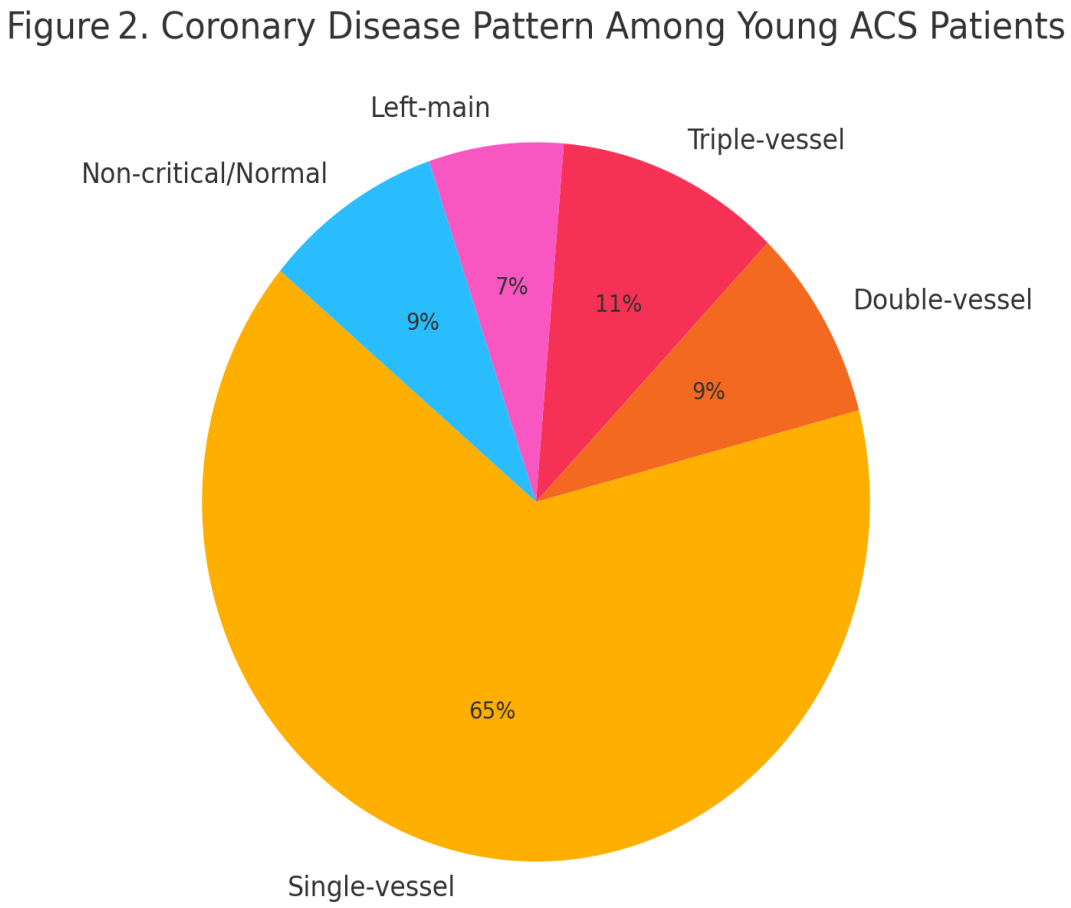

Figure 2. Coronary Vessel-Disease Pattern in Young ACS Patients *(pie chart showing the proportions of single-, double-, triple-vessel, left-main and non-critical/normal anatomy)*

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DISCUSSION

Young Indian adults who experience acute coronary syndrome (ACS) appear to sit at the epicentre of multiple converging epidemics—tobacco, visceral adiposity, dysglycaemia and low-grade systemic inflammation—and the present study offers one of the clearest high-resolution snapshots of how these forces jointly accelerate coronary disease before the age of forty-five. Half of our patients were active smokers, more than nine in ten derived at least five percent of daily calories from saturated fat, none achieved guideline physical-activity targets and two thirds held non-sedentary jobs that rarely translate into structured exercise; these numbers echo national surveys of urban youth but stand in stark contrast to Western registries, where lifestyle risks have slowly receded in the past decade [13, 14]. Equally striking was the burden of adiposity-related metabolic derangement: body-mass-index readings averaged 26.5 $kg\,m^{-2}$, yet fifty-four percent were frankly obese by Asian cut-offs and an additional fifteen percent overweight, and routine HbA1c screening unmasked occult diabetes in almost one third of patients who had never been labelled diabetic. These data reinforce the notion of a "South-Asian phenotype"—central adiposity, insulin resistance and atherogenic dyslipidaemia manifesting at lower BMI thresholds—that has long been implicated in premature coronary disease [15, 16].

Beyond conventional risks, pervasive inflammation emerged as a unifying thread. High-sensitivity C-reactive protein (hsCRP) exceeded 1 $mg\,dl^{-1}$ in eighty-three percent of our cohort and, in multigroup comparisons, was the only variable that distinguished infarction (STEMI/NSTEMI) from unstable angina; fully ninety-five percent of STEMI/NSTEMI patients carried elevated hsCRP versus one quarter of UA cases, underscoring the intimate link between systemic inflammation, plaque vulnerability and transmural necrosis. Histopathologic studies of young coronary deaths have documented lipid-rich, macrophage-dense plaques with thin fibrous caps that rupture readily under inflammatory stress [17]; our angiographic finding of intracoronary thrombus in twenty-two percent of patients with raised hsCRP provides in-vivo clinical corroboration. Homocysteine and lipoprotein(a)—heritable pro-inflammatory, pro-thrombotic mediators—were elevated in nearly half and one third of participants, numbers consistent with Bangladeshi and Pakistani cohorts and far higher than Caucasian comparators [18, 19].

The interplay among visceral fat, insulin resistance and inflammation likely fuels this aggressive biology. Excess adipose tissue operates as an endocrine organ, secreting interleukin-6 and tumour-necrosis-factor-α that in turn stimulate hepatic CRP synthesis; insulin resistance amplifies free-fatty-acid flux, generates small dense low-density lipoprotein particles, impairs endothelial nitric-oxide production and tilts the coagulation balance toward thrombosis [20]. Smoking superimposes oxidative stress and platelet hyper-reactivity on this milieu, and air pollution, chronic infections and psychosocial stress—ubiquitous in a megacity like Mumbai—may provide additional inflammatory triggers. These synergistic insults help explain why, despite high obesity and inflammatory indices, angiography still showed predominantly single-vessel, proximal left-anterior-descending (LAD) disease: accelerated focal plaque rupture on an otherwise limited atherosclerotic canvas. Such anatomy confers both clinical advantage—culprit-lesion revascularisation often suffices to restore large myocardial territories—and prognostic warning, as it might lull clinicians into underestimating residual systemic risk.

The practical implications are manifold. First, tobacco cessation remains the most accessible lever; given the median decade-long lag between quitting and risk normalisation, interventions must begin in adolescence. Second, our data argue for earlier and broader cardiometabolic screening: almost one third of patients harboured previously unrecognised diabetes, and one in five had LDL-C ≥130 $mg\,dl^{-1}$ despite their youth. Current Indian guidelines trigger lipid or glucose testing at thirty years only with family history or obesity [21]; our findings support lowering that threshold and incorporating point-of-care HbA1c and non-fasting lipid profiles into college or workplace health checks. Third, the strong association between hsCRP and infarction phenotype resurrects interest in targeted anti-inflammatory therapy. While high-cost biologics such as canakinumab have proven benefit in post-MI patients with persistent inflammation [22], low-dose colchicine—currently under evaluation in large pragmatic trials—may offer a viable, inexpensive option in resource-limited settings. Fourth, the low prevalence of angiographic calcification suggests that coronary calcium scoring may be less informative in this age group;

computed-tomography angiography or positron-emission-tomography tracers that visualise arterial inflammation could better stratify truly high-risk youth, though cost-effectiveness studies are urgently needed [23].

Our investigation is strengthened by prospective enrolment, uniform biochemical profiling and blinded angiographic adjudication, but several limitations warrant acknowledgement. The sample size, curtailed by COVID-19–related declines in cardiac admissions, limits statistical power and precludes robust multivariable modelling; single-centre design raises the spectre of referral bias toward more severe cases; and follow-up was confined to the index hospitalisation, preventing insight into longer-term recurrence or mortality. Moreover, hsCRP, although a validated risk marker, can rise with subclinical infections; while no patient had clinically evident sepsis, we cannot completely exclude confounding. Finally, we did not measure novel indices such as epicardial fat volume or oxidised LDL, which might have further illuminated pathophysiology.

Notwithstanding these caveats, the study reinforces a compelling narrative: premature ACS in Indian adults is largely a creature of modifiable behaviours acting upon an adiposity-driven inflammatory substrate. Turning that narrative around will demand coordinated action—taxation and regulation to curb tobacco and trans-fat availability, urban planning that rewards active transport, community programmes embedding lifestyle literacy into school curricula, and primary-care algorithms that flag high-risk youth through simple, affordable tests. Randomised trials evaluating integrated packages—cessation support, weight-management, aggressive LDL-C lowering and low-dose anti-inflammatories—specifically in the eighteen-to-forty age band could furnish the evidence base to shift guidelines from reactive middle-age treatment to proactive young-adult prevention. Ultimately, bending the arc of India's coronary epidemic will require moving "upstream," neutralising risk trajectories before the first plaque even forms and well before the first myocardial cell is lost [24, 25].

CONCLUSION

Young adults with ACS in Mumbai display a "double burden" of traditional risks (smoking, obesity, dysglycaemia) and aggressive inflammatory milieu. Routine screening for hsCRP and HbA1c in high-risk youth, combined with tobacco cessation, dietary reform and structured physical activity, could avert a sizeable proportion of premature coronary events. Multicentric studies with larger sample sizes are warranted to validate these findings and guide national prevention strategies.

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