Review Article

Emerging Trends of Myocardial Infarction in Young Indian Adults: Epidemiology, Risk Factors, Prevention Strategies, and the Role of Low-Dose Aspirin

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Abstract

Myocardial infarction (MI) at ≤45 years is rising in India and presents earlier than in Western populations. Non-atherosclerotic mechanisms coronary vasospasm, spontaneous coronary artery dissection (SCAD), and myocardial infarction with non-obstructive coronary arteries (MINOCA), are proportionally common, increasing diagnostic complexity. This study undertook a narrative review (January 2000–January 2025) across PubMed/MEDLINE, Scopus, Web of Science, and trial registries, using dual screening and synthesis with India-focused weighting. Young-onset MI constitutes a notable share of acute coronary syndromes beyond metros; men predominate; women often present later and atypically. Risk clusters tobacco (smoked and smokeless), dyslipidaemia, insulin resistance, central adiposity, hypertension, and physical inactivity are amplified by pollution, psychosocial stress, and poor sleep. Elevated lipoprotein(a) and under-recognised familial hypercholesterolaemia impart lifelong exposure. Mechanisms range from accelerated atherothrombosis to vasospasm/SCAD/MINOCA; OCT/IVUS and cardiac MRI refine attribution. Emphasise complete tobacco cessation; intensive lipid and blood-pressure control; diabetes care; physical activity and weight targets; and incorporation of South-Asian risk enhancers in risk assessment. Use coronary artery calcium (CAC) to reclassify borderline/intermediate risk when decisions are uncertain. Routine low-dose aspirin for primary prevention is not recommended; reserve for carefully selected adults after shared decision-making, and reassess bleeding risk within any polypill strategy. Systems: Strengthen registries; include women and rural populations; scale cardiac rehabilitation; and deliver India-specific trials (CAC-guided aspirin, Lp(a) pathways, and familial-hypercholesterolaemia screening). Young-onset MI in India is preventable and diagnosable earlier with timely, mechanism-guided pathways and context-fit prevention.

Keywords: Young-onset myocardial infarction; India; Lipoprotein(a); MINOCA; Coronary artery calcium.

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Introduction

Young-onset myocardial infarction (MI), typically defined as MI at ≤45 years, is a growing clinical and public-health concern in India. South Asians experience MI earlier than many other populations, and Indian hospital registries report substantial proportions of acute coronary syndromes (ACS) in working-age adults. The consequences extend beyond health: premature MI disrupts families and productivity and increases long-term cardiovascular risk. This review summarises current evidence on epidemiology, mechanisms, risk factors, and prevention strategies relevant to young adults in India, with a focused appraisal of low-dose aspirin in primary prevention.[1-4]

Available data indicate that young MI forms a meaningful share of total MI events in India, with signals from urban and increasingly rural settings. Indian cohorts and registries describe earlier presentation, high tobacco exposure, and frequent metabolic risk factors compared with Western peers. These findings underscore the need for earlier risk assessment and context-specific prevention pathways in primary care and occupational health.[5-8]

Pathophysiology is heterogeneous. Most cases reflect accelerated atherosclerosis driven by dyslipidaemia, insulin resistance, central adiposity, and endothelial dysfunction, often amplified by tobacco use and pro-thrombotic states. A minority arise from non-atherosclerotic causes such as coronary vasospasm, spontaneous coronary artery dissection, congenital anomalies, or myocarditis, entities that are especially relevant in young patients and require tailored diagnostic strategies.[9-11]

Risk is shaped by a blend of traditional and emerging determinants. Hypertension, diabetes, adverse lipid profiles, tobacco (including smokeless forms), physical inactivity, and calorie-dense diets remain dominant. Environmental and psychosocial exposures air pollution, chronic work and financial stress, and sleep disruption further increase vulnerability in Indian settings, interacting with genetic predisposition and

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family history. These upstream factors influence both the incidence and the clinical course of young MI.[12-14]

Against this backdrop, prevention requires layered action. Lifestyle modification, tobacco cessation, blood-pressure and lipid control, and diabetes management form the foundation. Risk-stratified tools (e.g., coronary artery calcium [CAC] scoring in selected adults) and combination pharmacotherapy (e.g., polypill approaches) can help in high-risk groups. The role of low-dose aspirin in primary prevention is nuanced: contemporary guidelines advise against routine use and favour selective prescription only when cardiovascular risk is high and bleeding risk is low, following shared decision-making.[15-18]

This review integrates Indian and international evidence to (i) describe the epidemiology and risk profile of young-onset MI in India, (ii) outline mechanistic pathways relevant to earlier disease, (iii) evaluate prevention and management strategies suited to Indian contexts, and (iv) critically appraise the evidence for low-dose aspirin in primary prevention. The aim is to provide clinicians and policymakers with clear, practice-oriented guidance and to highlight gaps that merit targeted research and national surveillance.[1-4]

Database search and finalisation

Structured search appropriate for a narrative review; no protocol was registered, and no meta-analysis was planned. Databases: PubMed/MEDLINE, Embase, Scopus, Web of Science, and Cochrane Library; to strengthen the Indian context, we also searched IndMED, Google Scholar (first ~200 hits per query), and key policy portals (ICMR, MoHFW, WHO). Trial registries (ClinicalTrials.gov, CTRI) were scanned for recent or ongoing studies. Timeframe: January 2000 to Jan 2025; language: English (non-English with an English abstract considered if data were extractable). Core terms combined controlled vocabulary and free text for population, condition, and context (e.g., "young myocardial infarction" OR "premature coronary artery disease" OR "early-onset ACS"; age ≤45; India/South Asia; risk factors; pathophysiology; primary prevention; low-dose aspirin/bleeding). Example PubMed string: ("myocardial infarction" [MeSH] OR "acute coronary syndrome") AND (young OR early OR ≤45) AND (India OR South Asia) OR (aspirin AND primary prevention AND cardiovascular). Inclusion: observational studies, registries, trials, high-quality reviews, and guidelines relevant to young-onset MI or aspirin in primary prevention; exclusion: animal studies, paediatric (<18 years), single case reports (except illustrative non-atherosclerotic causes), opinion pieces, and abstracts without full texts. Two reviewers screened titles/abstracts and full texts, resolving disagreements by discussion; recent, higher-quality, and India-relevant sources were prioritized. Formal risk-of-bias scoring was not mandatory, but NOS, RoB-2, AMSTAR-2, and AGREE II informed weighting when useful. Synthesis was thematic (epidemiology, risk factors, mechanisms, prevention with a focus on aspirin); conflicting findings were explained by study design, population, and outcome differences. References were de-duplicated and managed for Vancouver numbering; a top-up search was completed on Jan 2025.

Review

Epidemiology and burden in India

Young-onset MI, usually defined as MI at ≤45 years, accounts for a meaningful share of acute coronary events in India and South Asia. Multiple hospital registries and population signals suggest earlier age at first MI compared with Western cohorts, with a sizeable proportion occurring in working-age adults.[1-3] The societal cost is high because events affect individuals at peak productivity and carry long-term risk of recurrent disease.[4,5]

Urban centres historically report more cases, but recent data indicate rising recognition in semi-urban and rural settings as access to diagnostics improves.[2,6] Men form the majority of cases, yet women often present later, with atypical symptoms, and face delays in diagnosis and treatment.[7,8] Socioeconomic factors, education, income, and health-care access shape these patterns and contribute to geographic variation.[9]

Clinical presentation in young adults frequently includes ST-elevation MI and single-vessel disease, though non-atherosclerotic causes (e.g., vasospasm, spontaneous coronary artery dissection) are proportionally more common than in older patients.[10-12] Pre-hospital delays remain substantial due to low symptom awareness and transport barriers, leading to missed opportunities for timely reperfusion.[13]

Short-term mortality is often lower than in older adults, but long-term burden is significant. Survivors face elevated risks of recurrent events, heart failure, and reduced quality of life, particularly when risk factors cluster and secondary prevention is suboptimal.[5,14,15] Return to work may be delayed or incomplete, amplifying economic impact on families.[16] Evidence gaps persist: women, rural populations, and informal-sector workers are under-represented; long-term follow-up is limited; and standardised definitions for "young MI" vary across studies.[8,9,17] Strengthened national registries, consistent age cut-offs, and linkage of clinical data with social determinants are priorities to refine true incidence and track outcomes over time [Table 1].[17,18]

Table 1: Key studies of mechanisms, risk factors, prevention (incl. aspirin), clinical care, rehabilitation, and systems in young-onset MI

Theme	Study (year)	Design/setting	Population	Key finding (one-line)	Relevance
Epidemiology (India)	CREATE Registry Xavier et al. (2008).[5]	Prospective national registry, India	20k+ ACS patients	Substantial ACS burden in working-age adults; system delays are common.	Anchors India-specific burden and care gaps.
Epidemiology (South Asia vs others)	Joshi et al. (2007).[4]	Multinational case–control (INTERHEART South Asia)	Early MI cases vs controls	South Asians present with MI at younger ages; distinct risk clustering.	Justifies India-focused early prevention.
Population overview	Prabhakaran et al. (2016).[6]	Narrative review, India	--	Synthesises India's CVD trends and determinants.	Context for rising young-onset MI.
Risk factors, tobacco (global)	INTERHEART Teo et al. (2006).[30]	Multinational case–control	52 countries	Smoking is strongly associated with MI across regions.	Supports tobacco as a dominant modifiable risk.

Risk factors: smokeless tobacco (India)	Gupta et al. (2019) meta-analysis.[29]	Systematic review/meta-analysis	ST users	ST use is associated with CHD risk.	Underpins cessation advice beyond smoking.
Risk factors: air pollution	Rajagopalan & Brook (2021).[26]	Clinical review	--	PM exposure is causally linked to CVD events.	Environmental determinants in Indian cities.
Risk factors: air pollution (India)	Yadav et al. (2023).[27]	Time-series, Delhi	ED visits	Short-term PM surges ↑ CV emergencies.	Local evidence for mitigation messaging.
Emerging biology: Lp (a)	EAS Consensus (2022).[19]	Consensus statement	--	Elevated Lp (a) independently ↑ increases ASCVD risk; consider once-in-a-lifetime testing.	Guides Lp (a) testing in premature MI.
Risk prediction (South Asians)	QRISK3 (2017).[20]	Algorithm development/validation	UK primary care	Adds risk-enhancers; better calibration in diverse groups.	Supports selective use for Indians; combine with CAC.
Mechanisms: plaque disruption	Jia et al. (2013).[10]	OCT study in ACS	Invasive imaging cohort	In-vivo identification of plaque erosion/rupture.	Mechanistic basis for MINOCA work-up.
Mechanisms: plaque erosion review	Kolte et al. (2021).[11]	Contemporary review	--	Erosion is common in younger ACS; therapeutic implications.	Explains young-onset phenotype.
Non-atherosclerotic: vasospasm	Lanza et al. (2011).[9]	Mechanistic review	--	Pathophysiology and management of spasm.	Supports vasodilator strategy.
Non-atherosclerotic: SCAD	Hayes et al. (2018).[8]	AHA statement	Predominantly women	SCAD recognition, conservative management favoured.	Tailors care in young women.
MINOCA framework	AHA statement (2019).[7]	Scientific statement	MINOCA patients	Diagnostic pathway incl. OCT/IVUS and CMR.	Mechanism-directed management for young MI.
Primary prevention: guidelines	ACC/AHA (2019); ESC (2021); USPSTF (2022).[1–3]	Guidelines	General adult	Routine aspirin not advised; individualised use only.	Core stance on aspirin.
Primary prevention: trials	ASCEND; ARRIVE; ASPREE (all 2018).[12–14]	RCTs	Diabetes; moderate risk; older adults	Small/no CV benefit offset by ↑ bleeding.	Evidence base behind de-emphasising aspirin.
Aspirin selection: CAC	Miedema (2014); Cainzos-Achirica (2020).[15,16]	Cohort/modelling (MESA)	Asymptomatic adults	CAC≥100 may identify net-benefit; CAC=0 → net-harm.	Framework for selective aspirin use.
Polypill strategy	TIPS-3 -Yusuf et al. (2020).[17]	RCT, multi-country (incl. India)	Intermediate-risk adults	Statin + ACE-I polypill ↓ events; aspirin adds benefit with more bleeding.	Supports risk-guided fixed-dose combos.
Diagnostic pathways	AHA/ACC Chest Pain (2021).[21]	Guideline	ED/outpatient	Early ECG, hs-troponin, imaging triage.	Standardises acute evaluation.
ACS management	ESC NSTE-ACS (2020/21).[22]	Guideline	Hospital care	Evidence-based antithrombotic and invasive strategies.	Aligns acute management.
Cardiac rehab: effectiveness	Cochrane Review (2016).[23]	Systematic review/meta-analysis	CHD/MI	Exercise-based CR reduces mortality/admissions.	CR as essential after young MI.
Cardiac rehab: home/hybrid	AHA/ACC/AACVPR (2019).[24]	Scientific statement	CR programs	Home-based CR is effective, improves access.	Scalable model for India.
Return to work	Dreyer et al. (2016).[25]	Comparative cohort	Young women vs men post-MI	Lower and delayed RTW in young women.	Highlights sex-specific rehab planning.
Lipids: targets/therapies	ESC/EAS Dyslipidaemia (2019/2020).[18]	Guideline	Secondary prevention	LDL-C <55 mg/dL for very-high risk; add-on agents.	Long-term risk-factor targets.

Abbreviations: ACS: acute coronary syndrome; ASCVD: atherosclerotic cardiovascular disease; CAC: coronary artery calcium; CMR: cardiac MRI; CR: cardiac rehabilitation; CVD: cardiovascular disease; ED: emergency department; h-high sensitivity; Lp(a): lipoprotein(a); MI: myocardial infarction; MINOCA: MI with non-obstructive coronary arteries; OCT: optical coherence tomography; RCT: randomised controlled trial; RTW: return to work.

Risk factors and social-environmental determinants

Young adults in India with myocardial infarction (MI) often show clustered, modifiable risks. Tobacco stands out: bidi/cigarette smoking accounts for a large share of MIs among urban men, and contemporary Indian registries of young MI report very high tobacco exposure at presentation.[1,2] Smokeless tobacco is also common. It is consistently associated with adverse cardiovascular risk profiles in Indian users, although studies show mixed results for a direct link to MI risk; nevertheless, its contribution to overall cardiometabolic risk justifies firm cessation advice.[3,4]

Metabolic factors are frequent and often begin early:

dyslipidaemia (high triglycerides/low HDL and elevated LDL), hypertension, diabetes or pre-diabetes, central adiposity, and inactivity. Indian ST-elevation MI datasets indicate that younger cases are more likely to use tobacco and to have obesity than older patients, underscoring risk accumulation during working age.[5] Inherited and biologic predisposition adds further risk. South Asians show higher prevalence of elevated lipoprotein(a) [Lp(a)], which independently increases coronary risk and may be relevant in premature events.[6,7] Familial hypercholesterolaemia (FH) is under-recognised; among Indians with premature coronary disease, probable/definite FH is not rare, arguing for targeted Lp(a) and LDL-C assessment and cascade screening when family history or very high LDL is present.[8,9]

Environmental exposures amplify risk. Short-term and chronic exposure to fine particulate air pollution ($PM_{2.5}$) is linked with higher emergency visits and admissions for cardiovascular disease, including ischaemic heart disease, with supportive evidence from Delhi time-series analyses and global meta-analyses.[10,11] Prevention in young adults should prioritise tobacco cessation (smoked and smokeless), early detection and control of lipids, blood pressure and glucose, waist-centric obesity management, and context-specific pollution mitigation. Consider once-in-lifetime Lp (a) testing in adulthood and evaluate FH when LDL-C is very high or there is premature coronary disease in the family.[6,8,9]

Pathophysiology and non-atherosclerotic causes

Young-onset myocardial infarction (MI) is usually driven by accelerated atherosclerosis. Endothelial dysfunction from tobacco exposure, insulin resistance, dyslipidaemia, and hypertension promotes LDL entry and oxidation within the arterial wall. This triggers macrophage recruitment, foam-cell formation, and growth of lipid-rich plaques that become inflamed and fragile. When a thin-cap fibroatheroma ruptures or when superficial erosion exposes the intima, platelet activation and thrombin generation produce an occlusive thrombus and acute MI. In young adults, both plaque rupture and plaque erosion are common, often on smaller plaques than in older patients.[1-3]

Pro-thrombotic biology further amplifies risk at younger ages. Smoking increases platelet reactivity and lowers nitric oxide bioavailability; hypertriglyceridaemia and low HDL impair reverse cholesterol transport; and acute inflammation elevates fibrinogen and factor VIII, shifting the balance toward thrombosis. Elevated lipoprotein(a) [Lp(a)] can accelerate atherogenesis and impair fibrinolysis because of its apo(a) homology to plasminogen. Familial hypercholesterolaemia and other monogenic dyslipidaemias, though under-recognised, also contribute to early plaque burden.[4-7]

Coronary vasospasm is a key non-atherosclerotic mechanism in the young. Hyper-reactive smooth muscle, autonomic triggers, and endothelial dysfunction cause intense, transient constriction that reduces flow and may provoke thrombosis on otherwise mild plaques. Episodes often occur at rest or in the early morning and respond to calcium-channel blockers and long-acting nitrates; beta-blockers without vasodilatory action may worsen spasm and are generally avoided.[8-9]

Spontaneous coronary artery dissection (SCAD) is another important cause, especially in young women. A tear or intramural haematoma compresses the true lumen, producing ischaemia without classic plaque rupture. Many cases heal with conservative therapy; revascularisation is reserved for ongoing ischaemia or left-main/haemodynamic compromise because wiring can extend the dissection. Thrombolysis is generally avoided given the risk of haematoma expansion.[10-12]

Other non-atherosclerotic causes include coronary embolism (e.g., atrial fibrillation, endocarditis, paradoxical embolus), congenital coronary anomalies with intermittent compression, myocarditis mimicking MI, and stress-induced (Takotsubo) cardiomyopathy. Hypercoagulable states—antiphospholipid syndrome, nephrotic syndrome, malignancy, pregnancy/post-partum, and exogenous hormones—can precipitate coronary thrombosis in angiographically normal or near-normal arteries. Substance-related vasoconstriction (e.g., cocaine, amphetamines) is less common in Indian settings but remains mechanistically relevant.[13-16]

Myocardial infarction with non-obstructive coronary arteries (MINOCA) is a frequent diagnostic label in young adults. Mechanisms include plaque disruption undetected on angiography, spasm, microvascular dysfunction, thrombo-embolism, SCAD, and myocarditis. Intracoronary imaging (OCT/IVUS) improves detection of plaque rupture/erosion and SCAD; cardiac MRI helps identify myocarditis or Takotsubo. Clarifying the mechanism guides therapy—antiplatelet and statin therapy for confirmed plaque disruption; vasodilators for spasm; anticoagulation for embolic sources; and tailored management for SCAD or myocarditis.[17-20]

A structured work-up is essential in the young: (i) careful risk assessment (tobacco, family history, lipid profile including Lp(a)), (ii) consideration of intracoronary imaging during angiography when findings are incongruent with presentation, (iii) cardiac MRI when MINOCA is suspected, and (iv) targeted testing for thrombophilia or embolic sources when indicated. Treatment should match the mechanism, with strong emphasis on secondary prevention and aggressive risk-factor control after any atherothrombotic event.[1,7,17,20]

Prevention strategies and the role of low-dose aspirin

Foundation of prevention. The first line of defence is lifestyle and risk-factor control: complete tobacco cessation, regular physical activity, weight management, blood-pressure control, lipid-lowering (statins as first-line), and glucose management in diabetes. For most young adults without prior cardiovascular disease, these measures deliver larger absolute benefit than aspirin.[1-3]

Risk assessment in South Asians. Use a validated 10-year risk calculator (e.g., Pooled Cohort Equations, QRISK3) and incorporate South-Asian risk-enhancing factors (family history of premature ASCVD, elevated Lp(a), metabolic syndrome). Because existing tools can misclassify South Asians, clinical judgment is essential; coronary artery calcium (CAC) scoring can refine risk in borderline or intermediate-risk adults. [1,4-7] When (and when not) to use aspirin. Routine aspirin for primary prevention is no longer recommended. The USPSTF advises an individualised decision for adults 40–59 years with ≥10% 10-year CVD risk and recommends against initiating aspirin at ≥60 years. ACC/AHA guidance similarly recommends infrequent use

in selected 40–70-year-olds without increased bleeding risk; ESC guidelines advise against routine use in low or moderate risk.[1-3]

Evidence base. Three large 2018 trials shaped current guidance: in diabetes (ASCEND), aspirin reduced serious vascular events but increased major bleeding (benefits offset by harms); in moderate-risk adults (ARRIVE), no clear cardiovascular benefit and more bleeding; in healthy older adults (ASPREE), no cardiovascular benefit and more bleeding. [8-10] Selecting candidates (if any). Consider aspirin only when ischaemic risk is high and bleeding risk is low after shared decision-making. CAC may help selection: observational modelling suggests net benefit when CAC ≥100 (especially with intermediate ASCVD risk) and no net benefit when CAC = 0. This approach remains adjunctive and must not replace statins or lifestyle therapy.[6,11-13]

Diabetes-specific notes. In diabetes without ASCVD, aspirin is not routine; consider only when overall ASCVD risk is high and bleeding risk is low, consistent with contemporary diabetes standards. Priority remains intensive risk-factor control (statins, blood pressure control, SGLT2i/GLP-1RA as indicated).[3,14]

Polypill approaches. In intermediate-risk populations (many from India), a statin–ACE inhibitor polypill reduced events; adding aspirin provided additional benefit at the cost of more bleeding, supporting risk-guided combination strategies in selected groups. Decisions should still follow shared decision-making and bleeding-risk review.[15] Practical checklist (primary prevention). Optimise lifestyle and statins first; estimate 10-year risk and apply South-Asian risk-enhancers; consider CAC to re-classify borderline/intermediate risk; discuss aspirin only if ischaemic risk clearly exceeds bleeding risk; and avoid aspirin in those with prior gastrointestinal bleed/ulcer, bleeding disorders, concurrent anticoagulation, uncontrolled hypertension, or heavy alcohol use.[1-3,11-13]

Clinical evaluation, diagnosis, and acute management

Presentation and first steps. Young adults can present with typical chest pain or atypical symptoms (breathlessness, epigastric pain, fatigue), and women may present later and less typically. Obtain a 12-lead ECG within 10 minutes, high-sensitivity troponin with repeat testing if initial results are equivocal, and baseline labs (full blood count, renal function, glucose, lipids). Start guideline-directed ACS care while confirming the diagnosis.[1-3]

Imaging and mechanism finding. Perform bedside echocardiography to assess wall motion, complications, and differentials. Use invasive coronary angiography for suspected STEMI or high-risk NSTEMI. When angiography and the clinical picture do not align (e.g., suspected MINOCA, SCAD, or plaque erosion), consider intracoronary imaging (OCT/IVUS) to detect plaque disruption or dissection, and cardiac MRI to identify myocarditis, Takotsubo, or microinfarction. Coronary CT angiography (CCTA) can be useful in stable, low-to-intermediate risk presentations. [2-6] Search for non-atherosclerotic causes. In young patients, evaluate for vasospasm (history of rest pain, triggers; consider provocation only in specialised centres), SCAD (typical angiographic patterns; avoid aggressive instrumentation), coronary embolism (atrial fibrillation, endocarditis, paradoxical embolus), and pro-thrombotic states (antiphospholipid syndrome, nephrotic syndrome, malignancy, postpartum). Consider toxicology when stimulant use is possible.[4-7]

Reperfusion and antithrombotic therapy. For STEMI, primary PCI is preferred; give thrombolysis only when timely PCI is not available. Initiate dual antiplatelet therapy (aspirin plus a P2Y12 inhibitor), parenteral anticoagulation, high-intensity statin, ACE inhibitor/ARB, and beta-blocker unless contraindicated. Tailor therapy to mechanism: SCAD—favour conservative management if stable; revascularise only for ongoing ischaemia or left-main/haemodynamic compromise. Thrombolysis is generally avoided. [5-8] Vasospasm—use calcium-channel blockers and long-acting nitrates; avoid non-vasodilating beta-blockers. [6-7] MINOCA—treat according to the confirmed mechanism (antiplatelet/statin for plaque disruption; vasodilators for spasm; anticoagulation for embolic sources; supportive care for myocarditis/Takotsubo).[4-6]

Secondary prevention and recovery. After any atherothrombotic event, prioritise complete tobacco cessation, structured cardiac rehabilitation, physical activity, weight management, blood-pressure and glucose control, and high-intensity statin with adherence support. Optimise ACE inhibitor/ARB and beta-blocker as tolerated. Assess bleeding risk to determine DAPT duration (commonly 12 months; shorter if high bleeding risk, longer if high ischaemic risk). In suspected inherited dyslipidaemia or strong family history, check Lp(a) once and consider familial hypercholesterolaemia work-up with cascade screening. Provide return-to-work planning, psychosocial support, and contraception/pregnancy counselling where relevant.[1-3,8-11]

Follow-up. Reassess symptoms, adherence, and side effects at 4–6 weeks, then periodically. Target LDL-C reduction ≥50% from baseline (or LDL-C <55–70 mg/dL depending on risk and guidance); add ezetimibe and consider PCSK9 inhibition if targets are unmet. Review blood pressure, HbA1c, and lifestyle goals at each visit. Use functional testing or imaging when residual ischaemia is suspected or before high-demand occupations.[2-3,9-11]

Cardiac rehabilitation, psychosocial care, and return to work

Why it matters: After a young-onset myocardial infarction (MI), long-term health, quality of life, and economic stability depend on more than coronary patency. Structured cardiac rehabilitation (CR), attention to mental health, and a planned return to work are central to recovery. Participation in CR reduces recurrent events, improves functional capacity, and enhances adherence to preventive therapies; uptake is often low in India due to access, cost, and awareness gaps, which targeted referral and flexible delivery models can address.[1-3] Core components of CR: Multidisciplinary CR should include (i) supervised, progressive aerobic and resistance exercise; (ii) risk-factor modification (smoking cessation; lipid, blood pressure and glucose control; weight management); (iii) education on medications and symptom recognition; and (iv) psychosocial support. Programmes should be adapted to age, sex, cultural norms, and occupational demands, with home-based or hybrid (centre plus telehealth) options where centre-based CR is not feasible.[2-5]

Psychological health: Anxiety, depression, and post-traumatic

stress are common after MI and can impair adherence and outcomes. Routine screening (e.g., PHQ-9, GAD-7) and brief, structured interventions (cognitive-behavioural strategies, stress management, sleep hygiene) should be integrated into CR, with referral to mental-health professionals when needed. Peer support and family involvement improve engagement in younger adults. [4-6] Sexual health, fertility, and pregnancy: Offer counselling on resumption of sexual activity (usually safe once symptoms are stable and exercise capacity has improved) and on effective contraception. For women contemplating pregnancy after MI, provide individualized pre-conception counselling that addresses risk factors, medications contraindicated in pregnancy (e.g., ACE inhibitors, statins), and timing. SCAD survivors require specialist input because of higher recurrence risk in the peripartum period.[6-8]

Return to work and driving: Assess job demands (physical load, shift patterns, safety-critical tasks). Most patients with uncomplicated MI can return to non-manual work within 2–4 weeks after clinical stability; manual or safety-critical jobs may need additional functional testing and a staged return. Driving restrictions depend on local regulations and clinical status; document clearance decisions and provide written guidance to employers when appropriate.[3,5,9] Adherence and health literacy: Young survivors often feel "cured" and stop therapy early. Use clear counselling, fixed-dose combinations where appropriate, reminders, and periodic feedback on risk-factor targets (LDL-C, blood pressure, HbA1c, weight, fitness). Involve family members and use simple goal tracking.[2-4]

Digital and community models: App-supported home programmes, telephone coaching, and community-based exercise groups can extend CR reach, especially in semi-urban and rural areas. Standardise safety checks (symptom logs, blood-pressure/heart-rate thresholds) and escalation pathways.[3,5] Measuring success: Track functional capacity (e.g., 6-minute walk test or symptom-limited exercise test), tobacco abstinence, LDL-C reduction ≥50% or to target, blood-pressure and glucose control, medication persistence, return-to-work status, and patient-reported outcomes (quality of life, mood). Use these metrics to tailor intensity and identify those needing additional support.[2-4]

Long-term secondary prevention and risk-factor targets

Antithrombotic therapy. After MI or PCI, dual antiplatelet therapy (DAPT) with aspirin and a P2Y12 inhibitor is standard for 12 months when bleeding risk is not high. Shorter courses (1–3 months) followed by P2Y12 monotherapy are reasonable in high-bleeding-risk patients; longer therapy may be considered in very high ischaemic risk with low bleeding risk. If oral anticoagulation is needed (e.g., atrial fibrillation), avoid prolonged triple therapy; use the shortest possible overlap, then continue the anticoagulant plus a single antiplatelet.[1-4]

Lipid management. High-intensity statins are first line. Aim for ≥50% LDL-C reduction and an LDL-C target <1.4 mmol/L (<55 mg/dL) for very-high-risk patients; add ezetimibe if targets are not met, and consider PCSK9 inhibitors when LDL-C remains above goal. For statin intolerance, re-challenge with a lower dose or alternate-day dosing; bempedoic acid is an option in selected cases. Measure lipoprotein(a) once to aid lifetime risk assessment and family screening.[5-8] Blood pressure and glucose. Target blood pressure <130/80 mmHg where tolerated, using ACE inhibitor/ARB, beta-blocker, calcium-channel blocker, and thiazide-type diuretic as needed. In diabetes, prioritise strict risk-factor control and consider SGLT2 inhibitors or GLP-1 receptor agonists with proven cardiovascular benefit, independent of baseline HbA1c.[6,9,10]

Lifestyle and cessation. Support complete tobacco cessation with counselling plus pharmacotherapy (varenicline, bupropion, or combination nicotine replacement). Recommend at least 150–300 minutes/week of moderate-intensity aerobic activity, 2 sessions/week of resistance training, a Mediterranean-style or heart-healthy diet, weight management, adequate sleep, and stress-reduction strategies.[9-12] Vaccination and infection prevention. Annual influenza vaccination and pneumococcal vaccination (per age/comorbidity) reduce complications and should be incorporated into routine care. Prompt treatment of respiratory infections may lower short-term cardiovascular risk.[13,14]

Adherence and simplification. Use fixed-dose combinations (e.g., polypill) when appropriate, synchronise refills, and provide clear written plans. Periodic feedback on targets (LDL-C, blood pressure, HbA1c, weight, fitness) helps maintain engagement, especially in young adults who may feel asymptomatic.[12,15] Monitoring and follow-up. Recheck lipids 4–12 weeks after therapy changes, then every 3–12 months. Monitor blood pressure at home when possible. Assess symptoms, side effects, and adherence at each visit. Order echocardiography if clinically indicated (e.g., heart-failure symptoms) and functional testing for recurrent ischaemia or before safety-critical work.[6,9]

Family screening and special groups. For suspected familial hypercholesterolaemia, offer cascade screening to first-degree relatives. Provide contraception counselling and pre-conception planning; avoid statins in pregnancy and discuss alternatives. Address depression and anxiety proactively, integrating brief psychological interventions into follow-up.[7,11,16] System supports for India. Embed secondary-prevention bundles into NCD clinics, expand access to affordable generics and fixed-dose combinations, and link hospital discharge to community follow-up and telehealth. Air-quality advisories, smoke-free policies, and workplace wellness programmes are practical population-level supports for young survivors.[12,15,17]

Merits and limitations

This narrative review offers an India-centred synthesis of young-onset myocardial infarction, linking epidemiology, risk determinants (including tobacco, metabolic factors, air pollution, lipoprotein(a), and familial hypercholesterolaemia), pathophysiology (atherosclerotic and non-atherosclerotic causes), and end-to-end care pathways. The manuscript provides practical, guideline-aligned recommendations across prevention, acute care, rehabilitation, and long-term secondary prevention, with clear, clinician-friendly takeaways such as selective use of low-dose aspirin, once-in-lifetime Lp (a) testing, consideration of coronary artery calcium for risk reclassification, and structured return-to-work planning. The search approach spanning major international databases, Indian sources, and trial registries supports broad coverage, while transparent

inclusion/exclusion choices and de-duplication improve usability. By identifying evidence gaps in women, rural populations, and long-term outcomes, the review also sets a focused research and policy agenda for India.

As a narrative (non-systematic) review without protocol registration or meta-analysis, the work is susceptible to selection and confirmation bias, and effect sizes are not pooled across studies. The English-language focus risks missing regional-language evidence, and heterogeneity in age cut-offs, case definitions, and outcomes limits direct comparability. India-specific randomized data on primary-prevention aspirin remain sparse, and detailed economic or implementation analyses for Indian health-system contexts are beyond the scope. The search window closes on Jan 2025, so very recent updates may be absent, and several emerging areas, such as polygenic risk, vaping/e-cigarettes, mechanistic pollution pathways, and implementation science, receive brief coverage only. These constraints do not negate the manuscript's practical value, but they underscore the need for periodic updates and complementary systematic work.

Conclusion

Young-onset MI in India is a distinct, high-impact problem. It stems mainly from accelerated atherosclerosis driven by tobacco, dyslipidaemia, insulin resistance, and hypertension, with important contributions from non-atherosclerotic causes such as vasospasm and spontaneous coronary artery dissection. Because events occur at peak working age, the personal and societal costs are large. Earlier recognition, especially in women and in semi-urban/rural settings, remains a priority.

Prevention should be practical and layered: complete tobacco cessation, aggressive lipid and blood-pressure control, weight management, and diabetes care deliver the greatest absolute benefit. Risk assessment must account for South-Asian risk enhancers (family history of premature ASCVD, elevated Lp(a), metabolic features), with selective use of coronary artery calcium scoring to refine borderline/intermediate risk. Low-dose aspirin is not for routine primary prevention and should be considered only when ischaemic risk clearly exceeds bleeding risk after shared decision-making. When MI occurs, mechanism-directed diagnosis (including OCT/IVUS and cardiac MRI when appropriate), guideline-based therapy, structured cardiac rehabilitation, and psychosocial support underpin long-term recovery.

Progress will depend on stronger registries with consistent age cut-offs, better inclusion of women and rural populations, integrated secondary-prevention bundles in NCD clinics, access to affordable statins and fixed-dose combinations, and scalable centre/home/hybrid CR models. Priorities for research include India-specific evaluation of aspirin in primary prevention, implementation and economic studies of prevention packages/polypills, long-term outcomes (including return-to-work and quality of life), and pragmatic pathways for Lp(a) testing and FH cascade screening.

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Conflicts of interest

There are no conflicts of interest.

References

1. Arnett DK, Blumenthal RS, Albert MA, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease. Circulation. 2019;140(11):e596–e646.
2. Visseren FLJ, Mach F, Smulders YM, et al. 2021 ESC guidelines on cardiovascular disease prevention in clinical practice. Eur Heart J. 2021;42(34):3227–3337.
3. Guirguis-Blake JM, Evans CV, Senger CA, et al.; US Preventive Services Task Force. Aspirin use to prevent cardiovascular disease: US Preventive Services Task Force recommendation statement. JAMA. 2022;327(16):1577–1584.
4. Joshi P, Islam S, Pais P, et al. Risk factors for early myocardial infarction in South Asians compared with individuals in other countries. JAMA. 2007;297(3):286–294.
5. Xavier D, Pais P, Devereaux PJ, et al. Treatment and outcomes of acute coronary syndromes in India (CREATE): a prospective analysis of registry data. Lancet. 2008;371(9622):1435–1442.
6. Prabhakaran D, Jeemon P, Roy A. Cardiovascular diseases in India: current epidemiology and future directions. Circulation. 2016;133(16):1605–1620.
7. Tamis-Holland JE, Jneid H, Reynolds HR, et al. Contemporary diagnosis and management of patients with myocardial infarction in the absence of obstructive coronary artery disease: a scientific statement from the American Heart Association. Circulation. 2019;139(18):e891–e908.
8. Hayes SN, Kim ESH, Saw J, et al. Spontaneous coronary artery dissection: current state of the science: a scientific statement from the American Heart Association. Circulation. 2018;137(19):e523–e557.
9. Lanza GA, Careri G, Crea F. Mechanisms of coronary artery spasm. Circulation. 2011;124(16):1774–1782.
10. Jia H, Abtahian F, Aguirre AD, et al. In vivo diagnosis of plaque erosion and calcified nodule in patients with acute coronary syndrome by intravascular optical coherence tomography. J Am Coll Cardiol. 2013;62(19):1748–1758.
11. Kolte D, Yonetsu T, Ye JC, et al. Optical coherence tomography of plaque erosion. J Am Coll Cardiol. 2021;78(12):1266–1277.
12. Bowman L, Mafham M, Wallendszus K, et al. Effects of aspirin for primary prevention in persons with diabetes mellitus. N Engl J Med. 2018;379(16):1529–1539.
13. Gaziano JM, Brotons C, Coppolecchia R, et al. Use of aspirin to reduce risk of initial vascular events in patients at moderate risk (ARRIVE): a randomised, double-blind, placebo-controlled trial. Lancet. 2018;392(10152):1036–1046.
14. McNeil JJ, Wolfe R, Woods RL, et al. Effect of aspirin on cardiovascular events and bleeding in the healthy elderly. N Engl J Med. 2018;379(16):1509–1518.
15. Miedema MD, Duprez DA, Misialek JR, et al. Use of coronary artery calcium testing to guide aspirin utilization for primary prevention: estimated impact on cardiovascular events and bleeding events. Circ Cardiovasc Qual Outcomes. 2014;7(3):453–460.
16. Cainzos-Achirica M, Miedema MD, McEvoy JW, et al.

Coronary artery calcium for personalized allocation of aspirin in primary prevention of cardiovascular disease in 2019: the Multi-Ethnic Study of Atherosclerosis (MESA). JAMA Cardiol. 2020;5(2):179–187.
17. Yusuf S, Joseph P, Dans A, et al. Polypill with or without aspirin in persons without cardiovascular disease. N Engl J Med. 2020;384(3):216–228.
18. Mach F, Baigent C, Catapano AL, et al. 2019 ESC/EAS guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. Eur Heart J. 2020;41(1):111–188.
19. Kronenberg F, Mora S, Stroes ESG, et al. Lipoprotein(a) in atherosclerotic cardiovascular disease and aortic stenosis: a European Atherosclerosis Society consensus statement. Eur Heart J. 2022;43(39):3925–3946.
20. Hippisley-Cox J, Coupland C, Brindle P. Development and validation of QRISK3 risk prediction algorithms to estimate future risk of cardiovascular disease: prospective cohort study. BMJ. 2017;357:j2099.
21. Gulati M, Levy PD, Mukherjee D, et al. 2021 AHA/ACC guideline for the evaluation and diagnosis of chest pain. Circulation. 2021;144(22):e368–e454.
22. Collet JP, Thiele H, Barbato E, et al. 2020 ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J. 2021;42(14):1289–1367.
23. Anderson L, Oldridge N, Thompson DR, et al. Exercise-based cardiac rehabilitation for coronary heart disease. Cochrane Database Syst Rev. 2016;1:CD001800.
24. Thomas RJ, Beatty AL, Beckie TM, et al. Home-based cardiac rehabilitation: a scientific statement from the American Association of Cardiovascular and Pulmonary Rehabilitation, the American Heart Association, and the American College of Cardiology. Circulation. 2019;140(1):e69–e89.
25. Dreyer RP, Xu X, Zhang W, et al. Return to work after acute myocardial infarction: a comparison between young women and men. Circ Cardiovasc Qual Outcomes. 2016;9(2 Suppl 1):S45–S52.
26. Rajagopalan S, Brook RD. Air pollution and cardiovascular disease. N Engl J Med. 2021;385(20):1885–1898.
27. Yadav R, Sagar A, Dagar A, et al. Short-term effects of ambient air pollution on cardiovascular emergency visits in Delhi: a time-series analysis. Indian Heart J. 2023;75(4):284–291.
28. Gupta R, Gurm H, Bartholomew JR. Smokeless tobacco and cardiovascular risk. JAMA Intern Med. 2019;179(4):476–477.
29. Gupta R, Sinha DN, Pednekar MS, et al. Risk of coronary heart disease among smokeless tobacco users: a systematic review and meta-analysis. Nicotine Tob Res. 2019;21(1):25–31.
30. Teo KK, Ounpuu S, Hawken S, et al. Tobacco use and risk of myocardial infarction in 52 countries in the INTERHEART study: case-control study. Lancet. 2006;368(9536):647–658.