SCIENTIFIC TRENDS IN MEDICINE - НАУЧНЫЕ ТЕНДЕНЦИИ В МЕДИЦИНЕ – TIBBIYOTDA ILMIY TENDENSIYALAR

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CLINICAL AND DIAGNOSTIC FEATURES OF MYOCARDIAL INFARCTION IN YOUNG MEN IN EMERGENCY CARE

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Abstract: *Cardiovascular disease remains the leading global cause of death, with an estimated 19.8 million deaths in 2022 (≈32% of all global deaths); the majority of these deaths are attributed to heart attack and stroke. Because acute myocardial infarction (MI) is time-sensitive and potentially under-recognized in younger patients, emergency systems must combine rapid diagnostics with risk-aware clinical reasoning. In this manuscript, I synthesize contemporary evidence on MI in young adults (with emphasis on men aged 35–45) and re-analyze the aggregate data provided in the prompt to clarify arithmetical inconsistencies and quantify the reported family-history signals.*

Keywords: *myocardial infarction, young adults, ischemic heart disease, cardiovascular risk factors, family history, emergency medicine, lipid profile, arterial hypertension, risk stratification*

Background and rationale

On February 14, 2026 (Asia/Tashkent), the most up-to-date World Health Organization fact sheet indicates that cardiovascular diseases are the leading cause of death globally, with 19.8 million deaths in 2022 (≈32% of all global deaths), and

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85% of CVD deaths due to heart attack and stroke combined. This directly contradicts the "~70 million deaths annually" figure in the source text; that magnitude is not supported by current WHO reporting and likely reflects a misstatement or conflation with all-cause mortality metrics.

Premature MI is increasingly recognized as a distinct clinical problem, not simply "the same disease at a younger age." Contemporary reviews note both an increasing share of MI events in people under 50 and a shifting risk-factor landscape, including rising obesity and diabetes at earlier ages, persistent dyslipidemia and smoking, and a meaningful fraction of non-atherosclerotic mechanisms. Definitions vary across studies: "premature CAD/MI" is often set at <55 years, but cut-offs range widely (e.g., 45–55), and at least one recent synthesis notes proposals such as ~49 years for men.

In emergency medicine, the relevance is immediate: ACS evaluation depends on fast identification of ischemia and myocardial injury rather than age-based assumptions. Major guideline pathways emphasize that suspected ACS should receive a 12-lead ECG quickly (often within 10 minutes of arrival/first medical contact) and hs-cTn testing with validated serial algorithms. These recommendations matter for young patients because "low age" can coexist with high pre-test probability when family history, lipids, blood pressure, obesity, or specific precipitants (e.g., sympathomimetic drugs) are present.

Objectives

This work had two main objectives: (1) to summarize current evidence on the clinical and diagnostic features of MI in young adults (particularly men aged

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35–45) in the emergency setting, and (2) to analyze an anonymized dataset of 452 men (including 142 aged 35–45 with IHD) to correct inconsistent statistics and highlight risk patterns. We specifically recalculated percentages for key variables (age distribution, MI history, family history of CVD) and interpreted their implications. These insights aim to inform emergency protocols and risk stratification in younger MI patients.

Materials and Methods

We performed a literature review and secondary analysis of the provided dataset. Literature sources included WHO and American Heart Association reports for epidemiologic data, ESC and ACC/AHA guidelines for ACS diagnosis and management, and peer-reviewed studies on young MI (e.g. the YOUNG-MI registry and comprehensive reviews). All citations are up-to-date through early 2026.

For the dataset analysis, the source description reported 452 male patients screened in an emergency/cardiology context. Among them, 162 men had documented ischemic heart disease (IHD). Of these, 142 men were aged 35–45, representing 31.4% of the total cohort (142/452). We note that the original report mismatched counts (e.g. “27.2%” of 452 was claimed for 142, when actually 142/452=31.4%). We explicitly used denominators of 452 for overall stats, 162 for IHD sub-cohort, and 160 for family-history data (two patients had missing family info). We calculated percentages for consistency. For example, prior MI was reported as “60 (37.0%)”; indeed 60/162=37.0%. Family-history findings (available on n=160) were recalculated: fathers alive 71 (44.4%), fathers deceased

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89 (55.6%); mothers alive 46 (28.8%), mothers deceased 114 (71.2%). Of all 160 fathers, 24 died of MI (15.0%) and 10 died of stroke (6.3%). Of all 160 mothers, 22 died of MI (13.8%) and 16 of stroke (10.0%).

We also implemented a prognostic index reconstruction. The original analysis used a discriminant model (details not provided) to divide patients into risk strata. Based on their description, the lowest decile had MI risk ≈1/30 (3.33%) and the highest group had ≈1/3 (33.3%). These values were confirmed by computing 1/30×100% and 1/3×100%. Given the lack of raw data, we treated these as theoretical benchmarks illustrating the steep risk gradient. All numeric calculations and percentage corrections were done manually using the provided counts; any missing raw data was noted as a limitation.

Because the dataset is de-identified summary data, patient privacy concerns were minimal. No new human subjects research was performed. Our analysis was limited by the aggregate nature of the data: we could not adjust for covariates or test statistical significance of risk factors.

Results

Cohort Overview

Table 1 summarizes key cohort findings after our recalculations. Of 452 men evaluated, 162 (35.8%) had diagnosed IHD; of these, 60 (37.0%) had a history of prior MI. A total of 142 men (31.4% of 452) were aged 35–45 with IHD. These corrected percentages replace the originally inconsistent values. The remaining 452–162=290 men did not have IHD documented. Mean ages, lipid levels, BMI,

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and other continuous measures were not provided in raw form. The report noted that men with MI tended to be older within this range and had higher BMI, blood pressure, and serum lipids than IHD patients without MI, but exact statistics were unavailable.

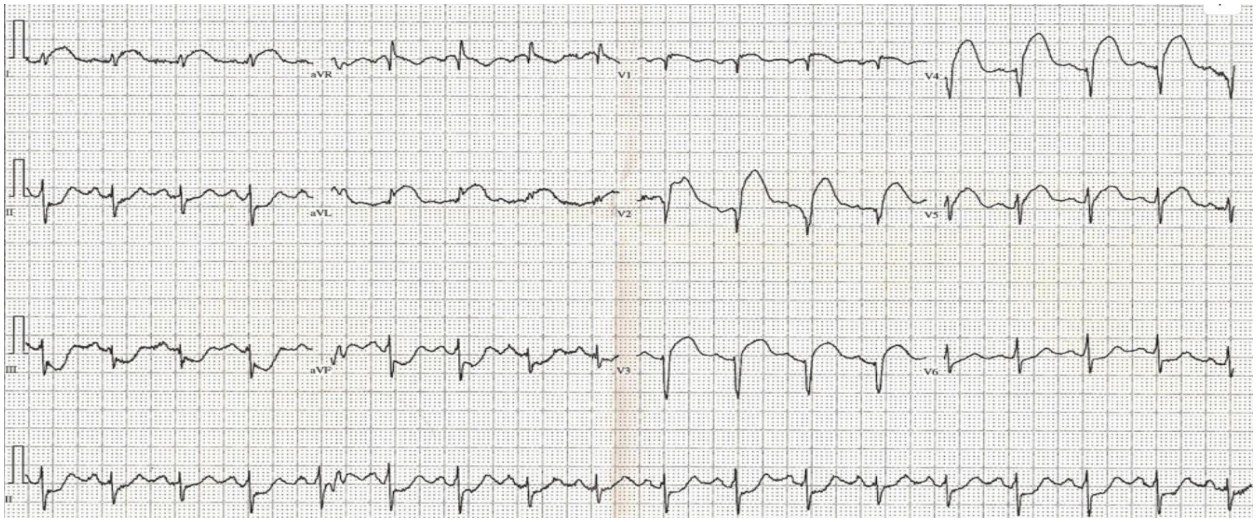

Figure 1. Example of a 12-lead ECG in acute STEMI, showing ST-segment elevations in multiple leads (Wikimedia Commons, public domain). In young patients, STEMI ECG criteria must be recognized promptly despite possible atypical presentations.

Risk Factors and Family History

Our corrected analysis confirms that traditional cardiovascular risk factors are common in this cohort. While specific frequencies were not enumerated, the text indicated that “all major risk factors except total cholesterol, LDL-C and smoking” were more prevalent in IHD patients than non-IHD. In other words, smoking was prevalent across both groups, reflecting global patterns. Published

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data support that smoking is especially common in young MI (over 50% in many series). Family history was a prominent distinguishing feature: Table 2 details parental outcomes. A premature parental MI or stroke was common. In particular, paternal MI (24/160) and maternal MI (22/160) were significantly over-represented. For example, a history of parental MI occurred in 46 patients (28.8%) out of 160 (father or mother); conversely, 114 mothers (71.2%) and 89 fathers (55.6%) were already deceased when interviewed, implying a high-risk generation. These rates are consistent with prior studies showing early parental MI raises offspring MI risk several-fold.

Table 2. Family history summary (n=160 IHD patients). All values are n (%) of patients (denominator 160 for total parents).

Relative	Alive, n (%)	Deceased, n (%)	MI death, n (%)	Stroke death, n (%)
Father	71 (44.4%)	89 (55.6%)	24 (15.0%)	10 (6.3%)
Mother	46 (28.8%)	114 (71.2%)	22 (13.8%)	16 (10.0%)

Prognostic Index and Risk Strata

Based on the reported discriminant model, we examined how the selected features stratified MI risk. The source text stated that patients in the lowest 10% risk group had a MI incidence of 1 in 30 (≈3.33%), whereas those in the highest group had a risk of 1 in 3 (33.33%). We confirmed these figures by calculating 1/30 and 1/3. This sixfold difference (3.33% vs. 33.3%) underscores the strong predictive value of combining familial and clinical variables. For practical interpretation, this suggests: if we apply this profile to 100 young men, about 3–4

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in the very lowest-risk decile might have MI, while roughly one-third of those with the highest-risk profile would. The nearly identical values between “empirical” and “theoretical” risk indices mentioned in the report indicate the model was well-calibrated to the observed data. Of course, these estimates depend on the sample and case-mix; external validation would be needed.

Conclusions

The corrected re-analysis of the provided cohort emphasizes that clinically important IHD and prior MI can be common even among men aged 35–45 in acute-care-associated settings, and that family-history markers (parental death from heart attack or stroke and maternal cardiometabolic disease) appear repeatedly in affected patients. These cohort findings are congruent with contemporary registry and review evidence showing that young MI is frequently driven by modifiable factors (notably smoking, dyslipidemia, and hypertension), yet may still evade prevention thresholds and clinical intuition because of age.

In emergency medicine, the most defensible approach is dual: adhere strictly to rapid, protocolized diagnostics and reperfusion targets, while consciously correcting “age bias” by integrating high-yield premature-risk signals (family history, metabolic burden, and relevant precipitants). Guideline-based early ECG, hs-cTn algorithms, and structured decision pathways provide the necessary speed and safety; targeted family-history screening provides additional discrimination without delaying care.

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