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ORIGINAL ARTICLES

Non-Conventional Risk Factors in Acute Coronary Syndrome

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ABSTRACT

Background: Acute coronary syndrome (ACS) remains a major cause of morbidity and mortality. Beyond conventional risk factors, non-conventional lifestyle and psychosocial exposures may contribute to adverse coronary and vascular phenotypes.

Objective: To evaluate the association of selected non-conventional exposures with ACS and to examine their relationships with structural vascular/adipose imaging markers.

Materials and Methods: We conducted a case–control study including 300 participants (150 ACS cases and 150 controls without ACS confirmed by coronary angiography or coronary CT angiography) at the University Hospital Center "Mother Teresa", Tirana (2024–2025). Physical activity, sleep duration, dietary pattern, alcohol use, and psychosocial factors were compared between groups, alongside epicardial adipose tissue (EAT), pericardial adipose tissue (PAT), and carotid intima–media thickness (CIMT; right and left).

Results: Cases were older than controls (66.1 ± 10.7 vs 57.4 ± 10.9 years) and more frequently male (74.7% vs 66.0%). Physical inactivity, short sleep duration, high-fat dietary patterns, and higher alcohol intake were more common among cases (all $p < 0.001$), whereas Mediterranean diet adherence was markedly lower. EAT and CIMT (right and left) were significantly higher in cases than controls (all $p < 0.001$), and PAT was also higher among cases ($p = 0.004$). Conclusions: In this Albanian cohort, ACS was strongly associated with modifiable non-conventional exposures—particularly short sleep, low physical activity, and unhealthy diet—and with adverse vascular/adipose imaging phenotypes. These findings support integrating key lifestyle and psychosocial factors alongside structural markers in regional cardiovascular risk profiling.

Keywords: acute coronary syndrome, sleep duration, physical activity, epicardial adipose tissue

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INTRODUCTION

Acute coronary syndrome (ACS)—unstable angina, non–ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI)—arises from acute plaque disruption or erosion with superimposed thrombosis and downstream myocardial ischemia (1).

Despite major advances in prevention and acute care, a substantial proportion of ACS risk and clinical heterogeneity is not fully captured by conventional risk factors alone, motivating broader, mechanistically grounded risk profiling (1,2).

In this context, non-conventional, modifiable exposures—short sleep duration, physical inactivity, unhealthy dietary patterns, harmful alcohol use, and psychosocial distress—have gained increasing epidemiologic and biological plausibility as upstream drivers of cardiometabolic dysregulation, autonomic imbalance, endothelial dysfunction, and vascular inflammation (2–4).

Large international data support meaningful contributions of lifestyle and psychosocial factors to myocardial infarction risk across diverse populations (4). Sleep, in particular, has shown consistent associations with incident myocardial infarction in contemporary cohorts, complementing guideline-level emphasis on sleep as a core component of cardiovascular health (2,5).

Parallel to exposure assessment, structural vascular and adipose phenotyping provides objective proxies of subclinical atherosclerotic burden and ectopic fat accumulation. Carotid intima–media thickness (CIMT) reflects subclinical atherosclerosis and improves prediction of future cardiovascular events in meta-analytic evidence (6).

Epicardial adipose tissue (EAT) represents a metabolically active visceral fat depot adjacent to the coronary arteries, capable of local paracrine pro-inflammatory signaling implicated in plaque vulnerability and adverse coronary phenotypes (7).

Perivascular adipose tissue has been proposed in CT-based studies as a sensor of vascular inflammation, including through metrics such as the perivascular fat attenuation index (FAI) (8, 9).

In the present study, adipose phenotyping was limited to EAT and pericardial adipose tissue. Accordingly, this study compares demographic characteristics and selected non-conventional exposures between ACS cases and angiography/coronary CT angiography–confirmed controls, and examines their relationships with imaging-based vascular/adipose phenotypes, including CIMT (right and left), EAT, and PAT.

MATERIALS AND METHODS

Study design and setting: Case–control study conducted at the Cardiology Service of the University Hospital Center "Mother Teresa", Tirana, Albania, during 2024–2025.

Participants: A total of 300 participants were enrolled. Cases (n=150) were hospitalized patients with confirmed ACS (STEMI, NSTEMI, or unstable angina), based on clinical criteria, ECG findings, and biochemical markers (including troponin when applicable). Controls (n=150) were individuals without ACS, in whom ACS/obstructive coronary disease was ruled out using coronary angiography or coronary CT angiography.

Variables: Demographics and anthropometrics included sex, age, body mass index (BMI), BMI categories, race, and residence (urban/rural). Non-conventional exposures included physical activity (high/moderate/low), sleep duration (<7 h/day, 7–9 h/day, >9 h/day), psychosocial profile (stress, anxiety, depression), dietary pattern (Mediterranean, high-fat, low-protein), and alcohol consumption (none, >1 drink/day, >2 drinks/day).

Statistical analysis

Categorical variables are presented as n (%) and continuous variables as mean ± SD. Between-group comparisons used the chi-square test (or Fisher's exact test when expected cell counts were <5) for categorical variables.

Continuous variables were compared using Welch's t-test for independent samples; Mann–Whitney U tests were used when distributional assumptions were uncertain. All tests were two-sided, and $p < 0.05$ was considered statistically significant. P-values are reported to three decimals, with $p < 0.001$ shown when applicable.

RESULTS

Baseline demographic characteristics of the study participants are presented in Table 1. Men predominated in both groups, with a higher proportion among cases than controls, and cases were notably older on average. Mean BMI and BMI-category distributions were broadly comparable between groups, with overweight and obesity common in both. Most participants were Caucasian, and urban residence predominated overall, although rural residence was proportionally more frequent among cases. BMI categories were calculated among participants with available height and weight measurements; missing values were excluded from the percentage denominators.

Lifestyle and psychosocial characteristics differed markedly between groups (Table 2). Physical activity levels were substantially lower among cases, with over half reporting low activity (56.0%) compared with only 13.3% of controls, while high activity was uncommon in cases (7.3%) but more frequent in controls (28.0%) ($p<0.001$). Sleep patterns also diverged strongly: short sleep (<7 h/day) predominated among cases (62.7%) but was rare among controls (11.3%), whereas the recommended 7–9 h/day category was reported by most controls (88.0%) and only about one-third of cases (36.0%) ($p<0.001$).

Dietary habits showed one of the largest contrasts. A Mediterranean dietary pattern was common in con-

trols (73.3%) but much less frequent in cases (20.0%), while a high-fat diet predominated among cases (76.0%) compared with controls (24.7%) (p<0.001). Alcohol consumption was also higher among cases: >1 drink/day was reported by 18.0% of cases versus 4.7% of controls, and >2 drinks/day by 6.7% versus 2.7%, respectively; abstinence was more common among controls (92.7%) than cases (75.3%) (p<0.001).

Table 1. Demographic characteristics of participants by group (Controls vs Cases)

Variable	Controls (n=150)	Cases (n=150)
Sex, n (%)		
Female	51 (34.0)	38 (25.3)
Male	99 (66.0)	112 (74.7)
Age, mean ± SD (years)	57.4 ± 10.9	66.1 ± 10.7
BMI, mean ± SD (kg/m²)	27.7 ± 3.5	27.4 ± 3.9
BMI categories, n (%)		
Underweight	0	2 (1.4)
Normal	27 (18.9)	33 (22.4)
Overweight	84 (58.7)	81 (55.1)
Obesity	32 (22.4)	31 (21.1)
Race, n (%)		
Caucasian	149 (99.3)	144 (96.0)
Mongoloid	0	2 (1.3)
Negroid	1 (0.7)	4 (2.7)
Residence, n (%)		
Rural	31 (20.7)	42 (28.0)
Urban	119 (79.3)	108 (72.0)

Across psychosocial variables, stress was highly prevalent in both groups (88.7% in controls and 90.0% in cases). Notably, depression was observed only among cases (4.7%), while anxiety was reported more frequently in controls (11.3%) than cases (5.3%) (overall p<0.001), suggesting that the between-group difference in the psychosocial domain was driven primarily by the presence of depression in cases and small shifts in anxiety distribution rather than by stress, which was near-universal in both groups.

Table 2. Lifestyle and psychosocial characteristics by study group

Variable	Controls n (%)	Cases n (%)	P
Physical activity			<0.001
High	42 (28.0)	11 (7.3)	
Moderate	88 (58.7)	55 (36.7)	
Low	20 (13.3)	84 (56.0)	
Sleep duration			<0.001
<7 h/day	17 (11.3)	94 (62.7)	
7–9 h/day	132 (88.0)	54 (36.0)	
>9 h/day	1 (0.7)	2 (1.3)	
Psychosocial factors			<0.001
Anxiety	17 (11.3)	8 (5.3)	
Depression	0	7 (4.7)	
Stress	133 (88.7)	135 (90.0)	
Dietary pattern			<0.001
Mediterranean diet	110 (73.3)	30 (20.0)	
High-fat diet	37 (24.7)	114 (76.0)	
Low-protein diet	3 (2.0)	6 (4.0)	
Alcohol consumption			<0.001
>1 drink/day	7 (4.7)	27 (18.0)	
>2 drinks/day	4 (2.7)	10 (6.7)	
Does not drink	139 (92.7)	113 (75.3)	

Figure 1 shows the distribution of EAT, PAT, and CIMT-R and CIMT-L in cases and controls. Across all four parameters, cases exhibit higher central tendencies than controls, indicating greater ectopic adipose tissue burden and increased carotid wall thickness in the ACS group.

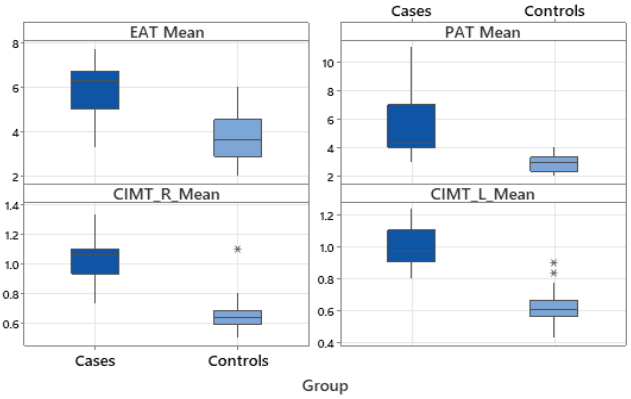

Figure 1. EAT, PAT, and Carotid Intima–Media Thickness by Study Group (Cases vs Controls)

DISCUSSION

This case–control study adds evidence that, in an Albanian clinical setting, non-conventional and modifiable exposures can meaningfully distinguish ACS cases from angiography/angio-CT confirmed controls, even when BMI distributions appear similar between groups. This pattern is consistent with the broader concept that acute coronary events reflect not only cumulative risk-factor burden, but also behavioral, autonomic, and inflammatory states that can promote plaque instability and thrombosis (1,2).

Sleep duration showed one of the strongest between-group contrasts, with a marked excess of short sleep among cases. The literature supports the biological plausibility and epidemiologic association of short sleep with adverse cardiometabolic profiles (blood pressure, insulin resistance), heightened sympathetic tone, and inflammatory activation—pathways relevant to ACS triggering and vulnerability (10).

Contemporary population evidence also suggests that adverse sleep and movement behaviors interact and jointly worsen cardiovascular risk trajectories, support-

ing the notion that sleep should be interpreted alongside lifestyle clustering rather than in isolation (4).

Similarly, the strong gradient observed for physical inactivity aligns with extensive evidence linking reduced activity to endothelial dysfunction and adverse vascular biology, and with prevention frameworks that treat physical activity as a core pillar of cardiovascular health (2,5).

From a mechanistic standpoint, the combination of low activity and short sleep plausibly amplifies autonomic imbalance, metabolic dysregulation, and inflammatory signaling—features increasingly recognized as relevant to coronary instability rather than only to long-term atherosclerotic progression (11).

The pronounced contrast in dietary pattern—high-fat patterns predominating among cases and Mediterranean diet adherence concentrated among controls—fits well with randomized and observational evidence supporting Mediterranean-style dietary patterns in cardiovascular prevention, likely mediated through lipid modulation, oxidative stress reduction, and anti-inflammatory effects (12).

In the global INTERHEART framework, dietary and psychosocial domains contribute materially to myocardial infarction risk across settings, reinforcing the relevance of incorporating these exposures in regional risk profiling (7).

Alcohol consumption was more frequent at higher daily levels among cases. Evidence on alcohol is complex and context-dependent; however, meta-analytic data indicate that alcohol intake can acutely increase cardiovascular event risk in the hours/days following consumption and that broader consumption patterns relate variably to CHD and stroke outcomes (13,14).

In an ACS-prone subgroup—particularly with comorbid hypertension, arrhythmogenic susceptibility, or metabolic risk—higher daily intake may therefore function more as a destabilizing co-factor than a benign habit (15).

The psychosocial findings warrant careful interpretation. Stress being near-universal in both groups suggests limited discriminatory capacity in this sample and may reflect measurement saturation or high background socioeconomic pressure. Nonetheless, psychosocial stress and depressed mood are consistently linked to both coronary disease development and acute triggering, with established pathways via neuroendocrine activation, inflammation, and behavioral mediation (16).

The observation that depression appeared only among cases could reflect pre-existing vulnerability, post-event recognition, or reverse causation; future work should clarify temporality with standardized tools and timing relative to the index event (10).

Together, these strands support the concept that non-conventional exposures and adverse vascular/adipose phenotypes may converge to shape ACS risk and clinical presentation, beyond what is captured by conventional factors alone (1,2).

CONCLUSION

In this case–control cohort, ACS was strongly associated with modifiable non-conventional exposures, particularly short sleep duration, low physical activity, unhealthy dietary patterns, and higher alcohol intake. In parallel, structural vascular/adipose imaging markers—EAT, PAT, and CIMT—were significantly higher in cases than in angiography/CT-confirmed controls, consistent with greater ectopic fat burden and increased subclinical atherosclerosis among cases.

These findings support incorporating key lifestyle and psychosocial factors alongside simple vascular/adipose imaging phenotypes in regional cardiovascular risk profiling. Prospective studies are needed to clarify temporality and to determine whether modifying these exposures improves imaging phenotypes and reduces ACS risk.

Editorial Statements

Ethics Approval and Consent to Participate
This study analyzed publicly available YouTube videos and did not involve human participants, patient data, or animal subjects. Therefore, according to institutional and international research ethics standards, formal ethical approval and informed consent were not required.

Data Availability Statement
The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request. All analyzed videos were publicly accessible on YouTube at the time of data collection.

Author Contributions (CRediT): Conceptualization & Methodology: TG, ER; Data Curation, Investigation and Writing – Original Draft: NSh: Formal Analysis: TG, SM: Writing – Review & Editing: TG, NSh: Supervision: TG. All authors have read and approved the final version of the manuscript.

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