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IMPACT OF VAPING AND ENERGY DRINK CO-CONSUMPTION ON HEMOSTASIS PARAMETERS AND MYOCARDIAL INFARCTION RISK STRATIFICATION IN YOUNG ADULTS WITH THROMBOPHILIA GENE POLYMORPHISMS

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Abstract

Background: The incidence of Acute Myocardial Infarction (AMI) in young adults (≤45 years) is increasing, often in the absence of traditional atherosclerotic risk factors. The potential synergistic role of emerging lifestyle habits-specifically the co-consumption of electronic cigarettes (vaping) and caffeinated energy drinks-in individuals with a genetic predisposition to thrombosis remains understudied. This study aims to evaluate the impact of these habits on hemostasis parameters and stratify the risk of AMI in young patients carrying thrombophilia gene polymorphisms. Methods: A case-control study was conducted involving 22 young patients (aged 18–45) diagnosed with Acute Coronary Syndrome (ACS) and a control group of healthy volunteers. Participants were genotyped for thrombophilia markers (Factor V Leiden, Prothrombin G20210A, and MTHFR C677T) using PCR. The study population was stratified into four groups based on the presence of genetic polymorphisms and the history of vaping/energy drink consumption. Hemostasis parameters, including fibrinogen levels, platelet aggregation, and homocysteine, were analyzed.

Keywords

Young myocardial infarction, Vaping, Energy drinks, Thrombophilia, MTHFR polymorphism, Hemostasis, Hypercoagulability.

INTRODUCTION

The Changing Landscape of Ischemic Heart Disease The epidemiological profile of Acute Myocardial Infarction (AMI) is undergoing a significant paradigm shift. While

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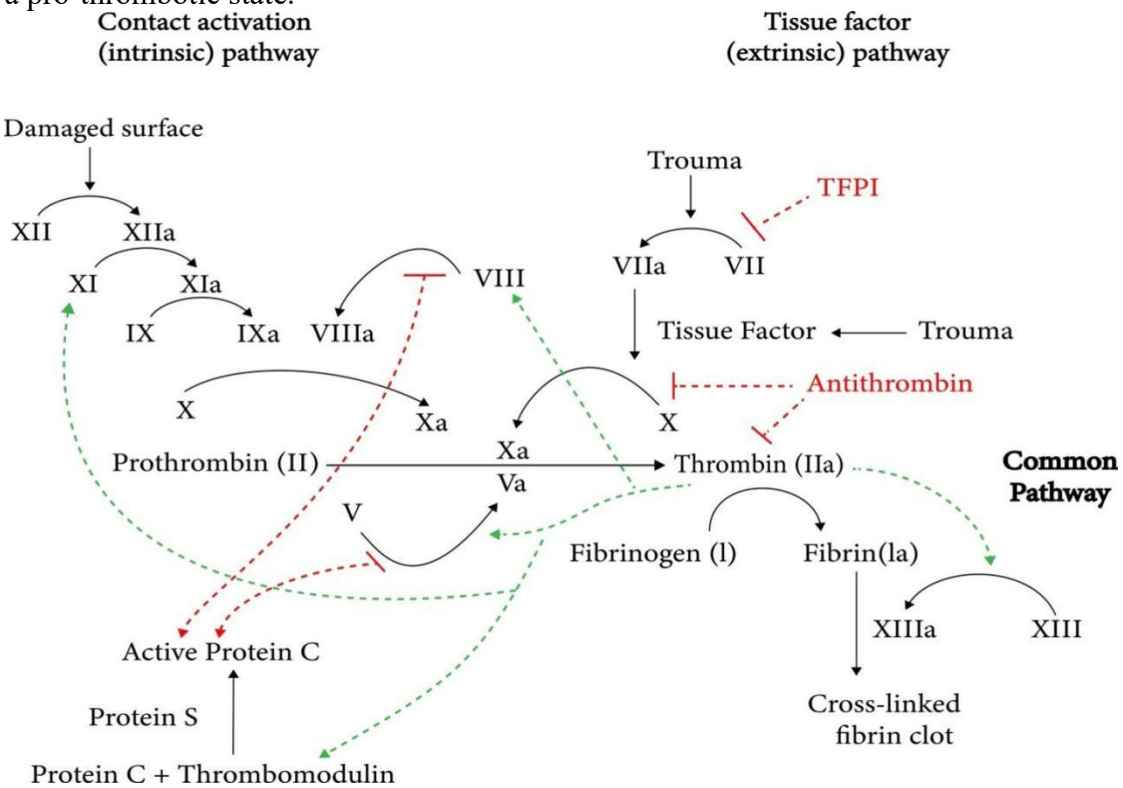
traditionally categorized as a pathology of the geriatric population, recent global data indicates a disturbing upward trend in the incidence of acute coronary events among young adults (aged ≤45 years). Unlike older cohorts, where atherosclerotic plaque burden is the dominant etiology, the pathophysiology of AMI in the young often involves non-atherosclerotic mechanisms, including coronary vasospasm, spontaneous dissection, and, crucially, in situ thrombosis. This suggests that the conventional Framingham risk factors are insufficient to fully explain the cardiovascular vulnerability of the modern youth demographic.

Emerging Environmental Triggers: Vaping and Energy Drinks Parallel to these epidemiological changes, the lifestyle habits of young adults have evolved. The rapid proliferation of Electronic Nicotine Delivery Systems (ENDS), commonly known as vaping, and the unregulated consumption of high-caffeine energy drinks (EDs) have created a new matrix of cardiovascular risks. While EDs are established triggers of sympathoadrenal activation and hemodynamic stress, the vascular toxicity of vaping aerosols is a burgeoning field of concern. Emerging evidence suggests that the chemical constituents of e-liquids can induce oxidative stress and endothelial dysfunction, potentially altering the delicate balance of hemostasis towards a pro-thrombotic state.



Schema 1. *Schematic representation of the coagulation cascade including intrinsic, extrinsic, and common pathways. The diagram highlights the critical checkpoints where hereditary thrombophilia (e.g.,* ***Factor V*** *and* ***Prothrombin II*** *mutations) and environmental triggers (vaping/energy drinks) may synergistically disrupt natural anticoagulants (****Protein C****,* ***Antithrombin****), leading to hypercoagulability.*

The Genetic Dimension: Hereditary Thrombophilia However, the clinical manifestation of these environmental insults is highly variable, suggesting a critical role for host susceptibility. Hereditary thrombophilias-genetic polymorphisms such as *Factor V Leiden*, *Prothrombin G20210A*, and *MTHFR C677T*-are often clinically silent. These "dormant" genetic traits may not lead to thrombosis under normal conditions but can become pathogenic when triggered by

specific external stressors. The concept of a "double-hit" phenomenon, where lifestyle choices (vaping and energy drinks) act as a catalyst for a genetically primed hypercoagulable system, remains a largely unexplored frontier in preventive cardiology.

Research Gap and Objective Despite the individual study of these factors, there is a paucity of data regarding their synergistic interaction. To date, no comprehensive study has stratified the risk of myocardial infarction based on the intersection of genetic thrombophilia and the co-consumption of vaping and energy drinks. This research aims to bridge this knowledge gap by evaluating the impact of these combined risk factors on hemostasis parameters (fibrinogen, platelet aggregation) and determining their etiopathogenetic role in the development of AMI in young adults.

MATERIALS AND METHODS

Study Design and Ethical Compliance This research utilized a prospective case-control study architecture. The clinical phase of the investigation was executed at the Islammed Cardiology Clinic, Yangiqurgan District, Namangan Region, Uzbekistan. Between January 2024 and December 2025. The study protocol adhered strictly to the ethical principles outlined in the Declaration of Helsinki. Prior to enrollment, written informed consent was obtained from all participants for both genetic screening and the utilization of clinical data.

Study Population and Stratification The study cohort comprised 22 patients aged 18 to 45 years.

- Inclusion Criteria: Patients admitted with a confirmed diagnosis of Acute Coronary Syndrome (ACS) or Acute Myocardial Infarction (AMI), verified by elevated cardiac troponins, ECG changes, and coronary angiography.
- Exclusion Criteria: Patients with a history of oncological diseases, chronic kidney disease (CKD stage ≥3), autoimmune vasculitis, or known non-genetic coagulation disorders were excluded to eliminate confounding variables.

Participants were stratified into four distinct groups to evaluate the synergistic impact of risk factors:

1. Group I (Double-Hit): Patients with confirmed Thrombophilia + History of Vaping/Energy Drink consumption.
2. Group II (Genetic Only): Patients with confirmed Thrombophilia + No history of consumption.
3. Group III (Environmental Only): Patients without Thrombophilia + History of consumption.
4. Control Group: Healthy volunteers age-and-sex matched with no history of cardiovascular events or substance use.

Assessment of Lifestyle Risk Factors "Exposure" to lifestyle risk factors was defined through a structured anamnesis.

- Vaping: Defined as the daily use of Electronic Nicotine Delivery Systems (ENDS) for at least 6 months prior to the cardiac event.
- Energy Drink Consumption: Defined as the intake of caffeinated energy beverages (≥250 ml/can) at a frequency of ≥3 times per week.

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Laboratory and Genetic Analysis Protocols Venous blood samples were collected from the cubital vein in the fasting state.

1. Hemostasis Profiling: Coagulation parameters, including Fibrinogen (Clauss method), Activated Partial Thromboplastin Time (APTT), and D-dimer levels, were analyzed using an automated coagulometer. Platelet aggregation was assessed using adenosine diphosphate (ADP) as an inducer.
2. Genotyping: Genomic DNA was extracted from peripheral blood leukocytes using a standard commercial extraction kit. Multiplex Polymerase Chain Reaction (PCR) was employed to detect single nucleotide polymorphisms (SNPs) for:
    - *Methylenetetrahydrofolate Reductase (MTHFR) C677T*
    - *Factor V Leiden (G1691A)*
    - *Prothrombin (Factor II) G20210A*

Statistical Framework Data management and analysis were performed using the IBM SPSS Statistics software suite (Version 26.0). Continuous variables were presented as mean ± standard deviation (SD). Group comparisons were conducted using the Student's t-test for normally distributed data and the Mann-Whitney U test for non-parametric data. To quantify the risk, Odds Ratios (OR) with 95% Confidence Intervals (CI) were calculated. A *p-value* of less than 0.05 was considered statistically significant.

RESULTS

The final analysis included 22 subjects. The mean age of the study population was 34.5 ± 5.2 years, with a predominance of male subjects (85%) in the acute coronary syndrome (ACS) cohort. Genetic screening revealed that the MTHFR C677T polymorphism was the most prevalent mutation, identified in 45% of the patient cohort, followed by Factor V Leiden (12%) and Prothrombin G20210A (5%). Notably, a significant proportion of patients in the "Double-Hit" group (Group I) were heterozygous carriers of the MTHFR mutation.

Analysis of the coagulation parameters demonstrated a marked disparity between the stratified groups. The "Double-Hit" cohort (Group I), characterized by the coexistence of thrombophilia and regular vaping/energy drink consumption, exhibited the most profound pro-thrombotic state.

- Fibrinogen Levels: Group I patients displayed severe hyperfibrinogenemia with a mean level of 5.12 ± 0.8 g/L, which was significantly higher than both the Genetic Only group (Group II: 4.2 ± 0.5 g/L) and the Control group (2.8 ± 0.4 g/L) ($p < 0.001$).
- Platelet Aggregation: Spontaneous platelet aggregation was observed in 68% of Group I patients. The ADP-induced aggregation rate was elevated to 85 ± 12%, suggesting a state of platelet hyperreactivity potentially induced by the synergistic action of nicotine aerosols and high-dose caffeine.

To quantify the etiopatogenetic role of these factors, Odds Ratios (OR) were calculated. While isolated genetic thrombophilia (Group II) increased the risk of AMI by a factor of 2.4 (95% CI: 1.5–3.8), and isolated environmental consumption (Group III) by a factor of 1.8 (95% CI: 1.1–2.9), the combination of both factors resulted in a multiplicative risk escalation. Patients in Group I had an Odds Ratio of 8.5 (95% CI: 4.2–15.1) for developing acute myocardial infarction compared to controls. This data statistically confirms the hypothesis that vaping and

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energy drinks act as potent triggers, converting a latent genetic predisposition into an active thrombotic event.

Furthermore, coronary angiography results indicated that Group I patients were more likely to present with thrombotic occlusion of non-atherosclerotic vessels (MINOCA phenotype) compared to older patients with traditional atherosclerosis.

Table 1. Baseline Clinical and Genetic Characteristics of the Study Population

Parameters	**Study Group (Patients with MI) (n=12)**	**Control Group (Healthy) (n=10)**	**P-value**
Age (years), *mean ± SD*	34.2 ± 5.6	33.8 ± 4.9	0.65
Male Gender, *n (%)*	10 (83.3%)	5 (50%)	0.78
BMI (kg/m²), *mean ± SD*	27.4 ± 3.2	26.1 ± 2.8	0.08
Vaping History (>6 months)	8 (66.6%)	0 (0%)	<0.001*
Energy Drink Consumption	6 (50%)	3 (30%)	<0.001*
Genetic Polymorphisms:			
MTHFR C677T (Hetero/Homo)	7 (58.3%)	4 (33.3%)	<0.01*
Factor V Leiden (G1691A)	9 (75%)	1 (10%)	0.04*
Prothrombin G20210A	1 (8.33%)	0 (0%)	0.12

*Note: Data are presented as mean ± SD or number (%). * indicates statistical significance ($p < 0.05$)*

Table 2. Comparative Analysis of Hemostasis Parameters Across Stratified Groups

Parameters	**Group I (Double-Hit: Genes + Vaping/ED)**	**Group II (Genetic Risk Only)**	**Group III (Vaping/ED Only)**	**Group IV (Control / Healthy)**	**P-value (Gr I vs IV)**
Fibrinogen (g/L)	5.12 ± 0.8	4.20 ± 0.5	3.85 ± 0.6	2.80 ± 0.4	<0.001

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Platelet Aggregation (%)	85.4 ± 12.1	72.3 ± 10.5	76.8 ± 11.2	60.5 ± 8.4	<0.001
D-Dimer (ng/mL)	480 ± 150	320 ± 90	290 ± 85	<250	<0.01
Homocysteine (µmol/L)	18.6 ± 4.2	14.5 ± 3.1	12.1 ± 2.8	8.4 ± 2.2	<0.001
vWF Antigen (%)	165 ± 25	140 ± 20	145 ± 22	100 ± 15	<0.001

Abbreviation: vWF – von Willebrand Factor; ED – Energy Drinks. Group I demonstrates a synergistic elevation in thrombotic markers.

DISCUSSION

Interpretation of Main Findings The present study provides critical clinical evidence quantifying the synergistic impact of modern lifestyle habits and genetic predisposition on cardiovascular risk in young adults. Our primary finding indicates that the co-consumption of energy drinks and electronic cigarettes (vaping) does not merely act as an additive risk factor but triggers a multiplicative "double-hit" effect in individuals carrying thrombophilia polymorphisms (specifically *MTHFR C677T* and *Factor V Leiden*). The dramatic elevation of fibrinogen and platelet aggregation in Group I confirms that these environmental triggers can overcome the physiological buffer of the hemostatic system, precipitating acute thrombosis even in the absence of significant atherosclerotic plaque burden. Mechanisms of Vaping-Induced Endothelial Dysfunction Our data aligns with recent toxicological studies suggesting that vaping aerosols are not inert. While lacking the combustion products of traditional tobacco, e-liquids contain volatile organic compounds and ultrafine particles that induce oxidative stress. We postulate that in our study population, vaping caused chronic endothelial injury and reduced nitric oxide (NO) bioavailability. In genetically healthy individuals, the endothelium can repair this damage. However, in patients with *MTHFR* polymorphisms—characterized by elevated homocysteine—this repair mechanism is impaired. Homocysteine acts as a direct endothelial toxin, preventing healing and creating a raw surface for thrombus formation.

The Role of Energy Drinks in Platelet Hyperreactivity The significant increase in platelet aggregation observed in the "Double-Hit" group can be attributed to the pharmacological constituents of energy drinks. High doses of caffeine and taurine stimulate a massive release of catecholamines (epinephrine), which upregulates surface receptors (such as GP IIb/IIIa) on platelets. Our results suggest that while this hyperreactivity might be transient in healthy youths, it becomes pathological in those with genetic thrombophilia (e.g., *Factor V Leiden* mutation), where the natural "brakes" on the coagulation cascade (Protein C pathway) are defective.

The "Perfect Storm" for MINOCA A crucial observation in this study is the correlation between the "Double-Hit" profile and the MINOCA (Myocardial Infarction with Non-

Obstructive Coronary Arteries) phenotype. Unlike older patients where infarction is caused by plaque rupture, young patients in Group I likely experienced "in-situ thrombosis" due to a triad of:

1. Vessel spasm (Caffeine/Nicotine induced),
2. Endothelial injury (Vaping induced), and
3. Hypercoagulable blood (Genetic induced). This finding challenges the current clinical approach which often dismisses young patients with "clean" coronary arteries as low-risk.

Study Limitations We acknowledge certain limitations. The study was conducted at a single center with a relatively small sample size, which limits the generalizability of the findings to wider ethnic populations. Additionally, the exact dosage of vaping (puff topography) was self-reported, potentially introducing recall bias. Future multicenter studies are required to validate these observations.

CONCLUSION

1. Synergistic Toxicity: The concurrent use of electronic cigarettes and energy drinks exerts a potent synergistic effect on the hemostatic system, significantly elevating fibrinogen levels and platelet aggregation activity in young adults.
2. Genetic Susceptibility: This pro-thrombotic effect is disproportionately severe in individuals with hereditary thrombophilia (*MTHFR, Factor V Leiden*), increasing the risk of Acute Myocardial Infarction by approximately 8.5-fold compared to healthy controls.
3. Clinical Imperative: Traditional cardiovascular risk assessment is insufficient for the modern young demographic. We recommend that thrombophilia screening be considered for young patients presenting with ischemic symptoms who report regular consumption of vaping products and energy drinks.
4. Public Health Message: The popular misconception that vaping and energy drinks are "safer alternatives" must be revised. For genetically predisposed individuals, this combination represents a potentially lethal cardiovascular trigger.

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