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Review

Myocardial Infarction After Energy Drink Overconsumption in Adolescents and Young Adults: Mechanistic Vulnerability in Strength Athletes, Extreme Dieters and Other High-Risk Groups

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ABSTRACT

Energy drink consumption is common among adolescents and young adults who use these products for perceived improvements in energy, alertness and athletic performance. Case reports and experimental studies suggest that high volume intake may precipitate myocardial infarction and related acute coronary syndromes, including in individuals without traditional cardiovascular risk factors. Available evidence supports several biologically plausible pathways, including sympathoadrenal activation with increased myocardial oxygen demand, impaired coronary perfusion reserve with vasomotor dysfunction, prothrombotic signalling through platelet activation and endothelial impairment and electrophysiologic instability with QTc prolongation and arrhythmia mediated ischemia.

This review synthesizes published cases and human physiologic studies and highlights subgroups with heightened vulnerability. These include strength athletes who use anabolic androgenic steroids or combine stimulants around training, individuals engaging in extreme dietary restriction or rapid weight cutting, patients with eating disorder behaviours that produce dehydration and electrolyte abnormalities and individuals exposed to sleep deprivation or heat stress that can intensify hemodynamic strain and hypercoagulability. Additional risk amplification may occur with alcohol or sympathomimetic co ingestion and in individuals with occult channelopathies or baseline endothelial susceptibility.

Current evidence suggests that energy drink overconsumption can act as an acute trigger for myocardial infarction in susceptible individuals. Prospective studies are needed to define exposure thresholds, clarify ingredient interactions and identify which populations are most at risk.

Keywords: Adolescents; Athletic performance; Sympathoadrenal activation

Introduction

Energy drink (ED) consumption is widespread among adolescents and young adults, driven by perceived benefits in alertness, energy and athletic performance. Concern has grown because EDs are often consumed in large volumes, labelling is inconsistent and regulatory oversight remains limited. Most formulations combine caffeine (often supplemented by guarana), taurine, sugars, B vitamins and botanical extracts, several of which have documented cardiovascular effects. Although moderate caffeine intake (≤400 mg/day) is generally considered safe, higher exposures can produce marked sympathoadrenal activation and hemodynamic stress. Because EDs vary widely in caffeine content (approximately 40–400 mg per serving) and are frequently consumed as multiple servings in a short period, dose stacking is common.

Since the late 1990s-beginning with Red Bull's U.S. launch in 1997-ED use has expanded rapidly, with global sales reaching $57 billion in 2020[1]. In the United States, EDs are among the most commonly used dietary supplements in youth. Nearly one-third of adolescents aged 12-17 report regular use[2], 51% of college students report at least monthly consumption and 45% of deployed military personnel report daily use[3].

Across published reports, ED exposure has been associated with myocardial infarction (MI), cardiac arrest, QTc prolongation, platelet activation, reduced myocardial perfusion, endothelial dysfunction, blood pressure elevation and altered cardiac contractility[4-9]. QT/QTc prolongation is clinically important because of its relationship to malignant ventricular arrhythmias, including torsades de pointes[10]. These findings support biologic plausibility for ED-associated acute coronary syndromes through converging pathways involving hemodynamic stress, impaired coronary perfusion, vasomotor dysfunction, prothrombotic signalling and electrophysiologic instability.

Given rising ED consumption among youth and the growing number of case reports describing MI in otherwise healthy individuals, we conducted a structured literature review of ED overuse and MI in adolescents and young adults (ages 13-32). Of these results there were 8 clinical studies and 7 case reports. Of these studies, 7 included young adults (aged 18-32), and 1 including an adolescent (age 13). We focus on cases without known cardiac disease to explore mechanistic vulnerabilities and to synthesize how ED-related physiologic changes may precipitate ischemia in susceptible individuals.

Methods

We performed a structured literature search in PubMed and Google Scholar from inception through January 2026 using combinations of "energy drink," "caffeine," "myocardial infarction," "acute coronary syndrome," "vasospasm," "platelet," "endothelial dysfunction," "QTc," "arrhythmia," "adolescents," and "young adults." Reference lists of key articles were screened for additional reports.

Eligible studies included case reports, case series, observational studies and human physiologic or mechanistic studies describing MI, ischemia, vasospasm, thrombosis, arrhythmias or troponin-positive acute coronary presentations temporally associated with energy drink exposure. We focused on individuals aged 13–32 years without known structural heart disease. Exclusion criteria were: non-English publications, studies outside the age range, absence of a temporal ED-event link or clear underlying cardiac disease.

The search yielded 214 records; 32 underwent full-text review and 8 clinical studies and 7 case reports. Of these studies, 7 included young adults (aged 18-32), and 1 including an adolescent (age 13).

Because most available evidence consists of case reports and small physiologic studies, causality cannot be inferred; however, consistent clinical patterns and convergent mechanistic data support biologic plausibility.

Energy Drink Composition and Cardiovascular-Active Ingredients

Energy drinks (EDs) typically contain caffeine (often supplemented by guarana), taurine, sugars, B vitamins and botanical extracts such as yerba mate or Ginkgo biloba. Although each ingredient has distinct physiologic effects, combined formulations may amplify cardiovascular stress, particularly when consumed rapidly or in large volumes. Many ED containers include multiple servings, yet are commonly consumed in a single sitting.

Caffeine

Caffeine is the primary stimulant in EDs and acts mainly through adenosine receptor antagonism, increasing alertness by reducing adenosine-mediated sleep signaling[11]. Because adenosine contributes to basal vasodilatory tone, its blockade can shift vascular balance toward vasoconstriction and reduced coronary perfusion reserve. Caffeine also increases sympathetic activity, circulating catecholamines, peripheral vascular resistance and renin release[12]. Through competitive phosphodiesterase inhibition, caffeine elevates intracellular cAMP, producing positive inotropy and increasing myocardial oxygen demand[13]. Importantly, caffeine can blunt adenosine-induced coronary hyperemia, as demonstrated in a study of 47 patients undergoing fractional flow reserve testing[14], suggesting reduced coronary vasodilatory reserve during physiologic stress.

Taurine

Taurine is a conditionally essential amino acid concentrated in excitable tissues, including myocardium. It contributes to osmoregulation, antioxidant defenses and modulation of ion fluxes[15]. Short-term taurine exposure can increase intracellular sodium via the taurine-sodium cotransporter, promoting calcium influx through the sodium-calcium exchanger and transiently increasing intracellular calcium in cardiomyocytes and vascular smooth muscle. Longer-term exposure may reduce calcium overload, indicating time-dependent effects[16]. In ED formulations, taurine may interact with caffeine to influence inotropy, chronotropy and electrophysiologic stability, particularly at high doses.

Sugars and metabolic effects

Many EDs contain substantial quantities of added sugars. Acute sugar intake can increase heart rate, cardiac output and blood pressure[18], adding metabolic and sympathetic stress to stimulant-mediated cardiovascular effects. Chronic high sugar intake is associated with obesity, diabetes and cardiovascular disease[17], though these long-term risks are less relevant to acute ED-associated events.

B-vitamin formulations

EDs frequently include high doses of B vitamins-thiamin (B1), riboflavin (B2), niacin (B3), pantothenic acid (B5), pyridoxine (B6), biotin (B7), folate (B9) and cobalamin (B12)-as cofactors in energy metabolism[19]. While deficiencies can impair metabolic pathways, most individuals meet daily requirements through diet. Repeated high-dose exposure via ED overconsumption is typically unnecessary and may contribute to metabolic strain in some contexts.

Botanical stimulants (Guarana and yerba mate)

Guarana and yerba mate are botanical stimulants containing caffeine and other methylxanthines[20]. Their inclusion can substantially increase total stimulant burden beyond labeled caffeine content, as manufacturers often list only added caffeine, not caffeine from botanical sources.

Ginkgo biloba

Ginkgo biloba is included in some ED formulations for purported cognitive benefits. It has platelet-activating factor antagonistic properties and case reports describe clinically significant bleeding associated with ginkgo use[22]. Although bleeding is the most recognized concern, ginkgo's vascular and platelet effects may interact with other ED constituents in ways that influence vascular tone and hemostasis.

Mechanistic Pathways Linking Energy Drinks to Myocardial Infarction

MI in adolescents and young adults associated with energy drink (ED) consumption is unlikely to arise from a single pathway. Instead, available evidence supports a convergent model in which acute sympathetic stimulation, coronary vasomotor instability, prothrombotic signaling, endothelial dysfunction and electrophysiologic disturbances interact to create conditions favorable for ischemia, thrombosis or dissection in susceptible individuals[3,10,13,23].

Sympathoadrenal activation and hemodynamic stress

Caffeine-mediated antagonism of adenosine receptors increases sympathetic tone, circulating catecholamines, heart rate, blood pressure and peripheral vascular resistance[11,12]. Through downstream effects on cyclic nucleotides, EDs can also increase inotropy, raising myocardial oxygen demand[13]. When consumed in high volumes or rapidly, these hemodynamic changes may produce demand ischemia even in the absence of fixed coronary disease, particularly during exertion, dehydration or sleep deprivation.

Coronary vasomotor dysfunction and reduced perfusion reserve

Adenosine plays a central role in coronary vasodilation and hyperemic reserve. By blocking adenosine receptors, caffeine can blunt adenosine-induced hyperemia, as demonstrated in patients undergoing fractional flow reserve assessment[14]. Reduced vasodilatory reserve, combined with tachycardia and elevated afterload, may tip the balance toward ischemia. This mechanism aligns with case reports describing coronary vasospasm following ED exposure[13].

Platelet Activation, endothelial dysfunction and hypercoagulability

Human studies consistently demonstrate acute prothrombotic physiology after ED ingestion. Worthley et al. found increased platelet aggregation and reduced endothelial function 90 minutes after consumption of a 250 mL ED in healthy volunteers[9]. Pommerening, et al. reported increased hypercoagulability on thromboelastography and heightened platelet activity via arachidonic acid pathways within approximately one hour of ED ingestion[31]. These findings provide a mechanistic bridge between ED exposure and coronary thrombosis, particularly in settings of dehydration androgenic steroid use or concurrent stimulant intake.

Electrophysiologic instability and arrhythmia-mediated ischemia

EDs have been associated with QT/QTc prolongation and a randomized controlled trial demonstrated greater QTc prolongation after ED consumption than after caffeine-matched controls, suggesting contributions from non-caffeine constituents[24]. Experimental work also indicates that caffeine–taurine combinations can facilitate ventricular arrhythmias in susceptible models[25]. Even without torsades de pointes, tachyarrhythmias can worsen supply-demand mismatch and precipitate ischemia. Individuals with electrolyte abnormalities, congenital channelopathies or stimulant co-exposures may be particularly vulnerable[10].

Spontaneous coronary artery dissection and vascular vulnerability

Spontaneous coronary artery dissection (SCAD) is rare in pediatrics but has been reported after ED consumption in an adolescent without classic risk factors[29]. Proposed mechanisms include catecholamine surges, platelet activation and circadian vulnerability. This aligns with a broader framework in which ED-induced sympathetic activation and vascular dysfunction can act as triggers in structurally susceptible vessels, even in the absence of atherosclerosis.

Results

Case reports of energy drink-associated myocardial infarction

Across the 10 identified case reports and small case series (~15 patients), otherwise healthy adolescents and young adults developed acute myocardial infarction (MI) or MI-like presentations shortly after consuming large quantities of energy drinks (EDs). Reported exposures ranged from first-time ED use preceding STEMI with spontaneous coronary artery dissection (SCAD) in a 13-year-old boy[29] to sustained high-volume intake such as 7-9 cans per day for one week in a young adult male who developed NSTEMI[33]. Another early report described a 19-year-old male with STEMI and a troponin I of 34.7 µg/mL after consuming 2-3 cans of Red Bull daily for one week[4].

Coronary angiography was frequently normal, suggesting functional mechanisms such as vasospasm or transient thrombosis rather than fixed atherosclerotic disease[4,7,13,29]. When abnormalities were present, they typically involved focal thrombus, such as left main and proximal LAD thrombus described by Ünal, et al.[7]. Several reports explicitly described dose stacking, rapid consumption or co-exposures (e.g., alcohol, stimulants), which may have amplified physiologic stress[7,13,31-33].

Angiographic findings and clinical presentations

Six human studies evaluated acute cardiovascular or hematologic effects of ED consumption in healthy young adults. Hemodynamic studies consistently demonstrated increases in systolic blood pressure and heart rate after ingestion of 250-500 mL of commercial EDs[3,30]. One randomized crossover trial showed reduced cerebral blood flow velocity compared with placebo[30].

Electrophysiologic studies found QT/QTc prolongation after ED intake, with one controlled comparison showing greater QTc prolongation than an equivalent caffeine dose, suggesting contributions from non-caffeine ingredients[24]. Hemostatic studies demonstrated increased platelet aggregation and reduced endothelial function within 90 minutes of ED ingestion[9], as well as increased hypercoagulability on thromboelastography with heightened platelet activity via arachidonic acid pathways within approximately 1 hour[31].

Patterns of energy drink exposure and dose stacking

Across case reports and physiologic studies, several recurring patterns emerged:

- High-volume or rapid ED intake preceded nearly all acute coronary presentations[4,7,29,31-33].
- Normal or near-normal coronary arteries were common, implicating vasospasm, transient thrombosis or supply-demand mismatch rather than atherosclerosis[4,7,13,29].
- Prothrombotic physiology-including platelet activation and hypercoagulability—was consistently demonstrated in controlled human studies[9,31].
- QTc prolongation and tachyarrhythmias appeared more pronounced with ED formulations than with caffeine alone[10,24].
- Co-exposures (alcohol, stimulants, anabolic-androgenic steroids, dehydration, sleep deprivation) were frequently present and may have amplified vulnerability[7,13,31-33].

Collectively, these findings indicate that ED-associated acute coronary events in young individuals occur in the setting of high-dose stimulant exposure, acute hemodynamic stress and transient vascular or electrophysiologic instability, rather than chronic coronary disease.

Case reports and angiographic patterns

The earliest published case report linking high energy drink (ED) consumption with myocardial infarction (MI) was described by Scott, et al.[4]. A 19-year-old male presented with acute chest pain, ECG findings consistent with STEMI and marked troponin I elevation (34.7 µg/mL; reference <0.07 µg/mL), supporting the diagnosis of MI. He had no traditional coronary risk factors, did not smoke and denied illicit drug use. Coronary angiography demonstrated normal coronary arteries. The event was attributed to reported intake of 2 to 3 cans of Red Bull daily for one week before presentation, with the authors proposing ED-associated ischemia as a plausible precipitant[4].

Polat et al. reported a second case involving a 13-year-old boy who developed acute crushing substernal chest pain and was diagnosed with STEMI associated with spontaneous coronary artery dissection (SCAD) after consuming his first ED approximately 8 hours earlier[29]. Predisposing conditions commonly linked to SCAD, including connective tissue disorders and cocaine use, were reportedly excluded **(Table 1)**. The authors hypothesized that ED-related circadian disruption contributed to catecholamine and cortisol surges with increased platelet aggregation, potentially facilitating STEMI and SCAD in this adolescent[29].

Additional case reports describe acute coronary syndromes following very high daily ED intake. Wajih Ullah et al. reported NSTEMI in a young male after consumption of 7 to 9 cans per day for one week[33]. Ünal, et al. described STEMI with angiographic left main and proximal LAD thrombus in a young male after ED exposure[7]. Across reports, recurring patterns include angiographically normal coronaries, vasospasm and acute thrombosis, aligning with proposed ED-associated prothrombotic and vasomotor effects that may precipitate ischemia even in the absence of established coronary artery disease[7,13,31-33].

Table 1: Reported cases of myocardial infarction and acute coronary syndromes temporally associated with high-volume energy drink consumption in adolescents and young adults.

First author (year)	Age/sex	Reported ED exposure	Presentation	Peak troponin	Angiography / key finding	Proposed mechanism
Scott (2011)	19M	2–3 cans Red Bull daily for 1 week	STEMI	Troponin I 34.7 µg/mL (ref <0.07)	Normal coronary arteries	Functional ischemia trigger from high ED intake (sympathoadrenal stress, vasomotor dysfunction)
Polat (2013)	13M	First ED; symptoms ~8 hours later	STEMI	Not specified	SCAD	Catecholamine and cortisol surge with platelet aggregation, possibly linked to circadian disruption
Wajih Ullah (2018)	Young male	7–9 cans/day for 1 week	NSTEMI	Not specified	Not specified	ED-associated prothrombotic and vasomotor effects; dose stacking
Ünal (2015)	Young male	ED exposure (amount not specified in section)	STEMI	Not specified	Left main and proximal LAD thrombus	Acute thrombosis in the setting of ED-associated prothrombotic shift

Platelet aggregation, endothelial dysfunction and hypercoagulability (human studies)

Human studies support acute prothrombotic and vasoregulatory changes after energy drink (ED) ingestion in healthy young adults. In a randomized crossover study evaluating "Red Bull," consumption of a single ED increased blood pressure and heart rate compared with placebo and reduced cerebral blood flow velocity, suggesting short-term adverse hemodynamic and cerebrovascular effects[30]. Worthley, et al. assessed platelet and endothelial responses in 50 healthy individuals after 250 mL of an ED and observed increased platelet aggregation with reduced endothelial function 90 minutes post-consumption relative to baseline[9]. Together, these findings are consistent with a shift

toward thrombosis and impaired vasodilatory capacity.

Coagulation effects have also been demonstrated using viscoelastic testing. Pommerening et al. evaluated 24 healthy subjects after 500 mL of a commercial ED and found increased hypercoagulability on thromboelastography compared with baseline. Results also supported heightened platelet activity via arachidonic acid pathways within approximately 1 hour compared with water[31]. When considered alongside endothelial impairment, these changes provide a plausible physiologic bridge from ED exposure to coronary thrombosis, particularly in settings of dose stacking or concurrent risk modifiers.

Table 2: Human studies demonstrating platelet activation, endothelial dysfunction and hypercoagulability following energy drink consumption.

Study	Design / population	ED exposure	Comparator	Key measured endpoints
Grasser (2015)	Randomized crossover; young adults	Single "Red Bull"	Placebo	BP, heart rate, cerebral blood flow velocity
Worthley (2010)	Before-after in healthy volunteers (n=50)	250 mL ED	Baseline	Platelet aggregation; endothelial function
Pommerening (2015)	Before-after in healthy volunteers (n=24)	500 mL ED	Water and baseline	Thromboelastography; platelet activity (AA pathway)

Research Gaps and Future Directions

The available evidence suggests that energy drink overconsumption can act as an acute trigger for myocardial infarction in susceptible adolescents and young adults, but the current literature contains several important limitations that restrict causal inference. Most published cases involve single individuals and exposure quantification is often imprecise. Many reports lack detailed information on timing, co-ingestants, hydration status, sleep patterns or training load, all of which may influence physiologic response[4,7,13,29,31-33]. Controlled human studies provide valuable mechanistic insight, but sample sizes are small and exposure conditions do not reflect the high volume, rapid consumption patterns described in clinical cases[9,30,31].

Future research should prioritize prospective studies that examine dose thresholds, patterns of intake and interactions among caffeine, taurine, sugars and botanical stimulants. Ingredient interactions remain poorly understood, particularly regarding their combined effects on platelet activation, endothelial function and electrophysiologic stability[9,24,31]. Studies that compare commercial formulations with caffeine-matched controls are needed to clarify whether non-caffeine constituents meaningfully alter cardiovascular risk[24]. Additional work is also needed to determine whether repeated daily exposure produces cumulative physiologic effects that differ from single-dose studies.

Another critical gap involves the identification of vulnerable subgroups. Strength athletes using anabolic androgenic steroids, individuals with eating disorder behaviors and those with electrolyte abnormalities or congenital channelopathies may have reduced physiologic reserve[10,11,12,24,31]. These populations require targeted investigation to determine whether energy drink exposure produces exaggerated hemodynamic, prothrombotic or electrophysiologic responses. Research that incorporates real-world modifiers such as dehydration, sleep deprivation, heat stress and stimulant co-use would improve ecological validity and help clinicians better assess risk[7,13,31-33].

Finally, there is a need for standardized reporting of energy drink exposures in clinical settings. Emergency departments rarely document brand, volume, timing or co-ingestants, which limits the ability to identify patterns or establish temporal relationships. Improved surveillance and consistent reporting would support more accurate epidemiologic assessment and guide regulatory discussions regarding labeling, serving sizes and marketing practices directed at youth.

These research priorities are essential for clarifying the cardiovascular effects of energy drinks and for determining which individuals are most at risk for ischemic or arrhythmic complications. A more robust evidence base will allow clinicians to provide informed counseling and will support the development of targeted prevention strategies.

Discussion

Arrhythmia and ischemia in young energy drink consumers

In clinical practice, most young patients presenting after energy drink (ED) consumption report palpitations, tachycardia or anxiety, rather than chest pain. ED-related emergency visits frequently involve sinus tachycardia, supraventricular tachycardia, atrial fibrillation, ventricular ectopy or QT prolongation, even in otherwise healthy individuals[3,13]. These electrical disturbances reflect the potent sympathoadrenal stimulation produced by high-dose caffeine and other methylxanthines and are far more common than ischemic presentations.

Nevertheless, EDs can precipitate acute coronary vasospasm, platelet activation, hypertension and prothrombotic shifts, creating conditions under which myocardial infarction (MI) may occur in susceptible hosts[13,33]. EDs may also unmask congenital long-QT syndrome or exacerbate repolarization abnormalities, increasing the risk of tachyarrhythmias that can worsen supply-demand mismatch[3]. Thus, while arrhythmias represent the predominant ED-related cardiovascular presentation, clinicians should remain aware that ischemic events, though rare, are mechanistically plausible.

Across published reports, angiography in ED-associated MI typically reveals normal coronary arteries or focal thrombus, rather than diffuse atherosclerosis[4,7,13,29,31]. This pattern aligns with physiologic evidence demonstrating prothrombotic physiology, vasomotor dysfunction and electrophysiologic instability after high-volume ED intake[9,10,24,31-33].

Convergent mechanisms leading to myocardial infarction

A coherent mechanistic model emerges when ingredient level physiology is integrated with the clinical and physiologic evidence. Caffeine antagonizes adenosine receptors, which

increases sympathetic tone and circulating catecholamines while reducing vasodilatory signaling. These effects raise blood pressure, heart rate and myocardial oxygen demand[11,12]. Caffeine can also blunt adenosine mediated hyperemia, which implies reduced coronary perfusion reserve during physiologic stress[14].

Human studies show that energy drink exposure can increase platelet aggregation and impair endothelial function within ninety minutes, shifting vascular biology toward thrombosis and reduced vasodilatory capacity[9]. Additional work demonstrates increased hypercoagulability on thromboelastography, including evidence of heightened platelet activity through arachidonic acid pathways within approximately one hour[31]. These findings support a prothrombotic environment that may facilitate transient coronary obstruction in susceptible individuals.

Energy drinks have also been associated with QT and QTc prolongation and controlled comparisons indicate that formulations may prolong QTc more than caffeine matched controls[24]. Experimental studies suggest that caffeine and taurine combinations can promote ventricular arrhythmias in susceptible models[25]. Arrhythmias can worsen supply and demand mismatch and may precipitate ischemia in individuals with electrolyte abnormalities, inherited channelopathies or stimulant co-use[10].

Together, these processes create a physiologic environment characterized by increased myocardial oxygen demand, reduced coronary perfusion reserve, impaired endothelial function, heightened platelet activity and electrophysiologic instability. When intake is high or when vulnerability is present, these interacting pathways provide a plausible explanation for myocardial infarction or MI-like syndromes in adolescents and young adults without traditional coronary risk factors.

Populations with increased physiologic vulnerability

Strength athletes using anabolic-androgenic steroids: Strength athletes who use anabolic androgenic steroids often exhibit endothelial dysfunction, increased platelet reactivity, vasospasm, adverse lipid changes, myocardial hypertrophy and interstitial fibrosis. These changes reduce physiologic reserve and increase susceptibility to ischemic events. In this context, the sympathetic activation, impaired coronary perfusion and prothrombotic signaling associated with energy drink intake can act as acute triggers for ischemia or thrombosis[9,11,12,31]. Real world factors that are common among strength athletes, such as intense training, dehydration, sleep restriction and stimulant co-use, may further compound risk.

Individuals with extreme dieting or eating disorders: Eating-disorder physiology introduces several vulnerabilities relevant to ED exposure. Malnutrition can produce myocardial atrophy, conduction abnormalities and reduced autonomic reserve, while purging behaviors may lead to hypokalemia, hypomagnesemia and metabolic alkalosis, all of which heighten arrhythmia risk. Because EDs can increase heart rate and blood pressure and may prolong QT/QTc more than caffeine alone[10,24], individuals with electrolyte disturbances may have reduced repolarization reserve and increased susceptibility to malignant arrhythmias or arrhythmia-mediated ischemia. Rapid ED consumption during periods of caloric restriction may also exacerbate sympathetic activation and hemodynamic strain.

Athletes undergoing rapid weight cutting or heat stress: Athletes who engage in rapid weight cutting or who train in hot environments often experience dehydration, hemoconcentration and increased blood viscosity. These changes can amplify hypercoagulability. Energy drink associated platelet activation and endothelial dysfunction[9,31] may therefore have greater impact in these settings. Dehydration also potentiates tachycardia and reduces stroke volume, which increases the likelihood of supply and demand mismatch during stimulant exposure.

Sleep deprivation and circadian disruption: Sleep deprivation increases sympathetic tone, cortisol levels and platelet aggregability, creating a physiologic milieu that may interact with ED-related hemodynamic and electrophysiologic stress. Circadian vulnerability has been proposed in at least one case of ED-associated SCAD in an adolescent[29], suggesting that timing of intake may influence risk in susceptible individuals.

Individuals with occult channelopathies: ED-related QT/QTc prolongation, tachyarrhythmias and repolarization abnormalities may be particularly consequential in individuals with unrecognized congenital long-QT syndrome, Brugada syndrome or other channelopathies[10,24]. Even modest QTc prolongation may precipitate malignant arrhythmias in the presence of electrolyte abnormalities, stimulant co-use or rapid ED consumption. These individuals may also be more vulnerable to arrhythmia-mediated ischemia.

Baseline endothelial dysfunction and metabolic risk: Conditions associated with impaired endothelial function, such as smoking, early metabolic syndrome or inflammatory states, may amplify the platelet activation, endothelial dysfunction and hypercoagulability observed after energy drink intake[9,31]. In these individuals, the combination of sympathetic activation and reduced vasodilatory reserve may increase the likelihood of vasospasm or transient thrombosis.

Clinical implications for evaluation and counseling

ED exposure should be assessed explicitly in young patients presenting with chest pain, palpitations, syncope or otherwise unexplained troponin elevation. When ED-associated MI or MI-like syndromes are suspected, evaluation should consider vasospasm, thrombosis, SCAD, supply-demand mismatch and arrhythmia triggers. Counseling should not focus solely on caffeine content, as controlled comparisons suggest ED formulations may exert effects beyond caffeine alone, including QT/QTc changes[24]. Prevention messaging should also be targeted to higher-risk groups, including strength athletes using AAS, individuals with eating-disorder behaviors and those combining ED intake with intense exercise, dehydration or other stimulants[34-43].

Conclusion

Energy drink overconsumption has emerged as a plausible precipitant of myocardial infarction in adolescents and young adults who lack traditional cardiovascular risk factors. Across case reports and physiologic studies, a consistent pattern appears in which high volume or rapid intake produces acute sympathetic stimulation, reduced coronary perfusion reserve, platelet activation, endothelial dysfunction and electrophysiologic instability. These processes can interact to create conditions that favor ischemia, thrombosis or arrhythmia mediated injury in susceptible individuals.

The evidence base remains limited by small sample sizes, incomplete exposure characterization and a reliance on

single patient reports. Mechanistic studies demonstrate clear physiologic effects, but the relevance of these findings to real world consumption patterns is not fully understood. Vulnerable subgroups such as strength athletes using anabolic androgenic steroids, individuals with eating disorder behaviors and those with electrolyte abnormalities or congenital channelopathies may face amplified risk, yet these populations have not been systematically studied.

Clarifying dose thresholds, ingredient interactions and the influence of co-exposures such as dehydration, sleep deprivation and stimulant use will require prospective research with standardized reporting of energy drink intake. Improved surveillance and more rigorous study designs are essential for determining which individuals are most vulnerable and for guiding evidence based counseling. As consumption continues to rise among youth and young adults, a more comprehensive understanding of the cardiovascular effects of energy drinks is needed to inform prevention strategies and public health policy.

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